Transactions

of the

Forty-Fourth Annual Meeting

of the

American Academy of Ophthalmology and Otolaryngology

CHICAGO, ILLINOIS

October 8 to 13, 1939

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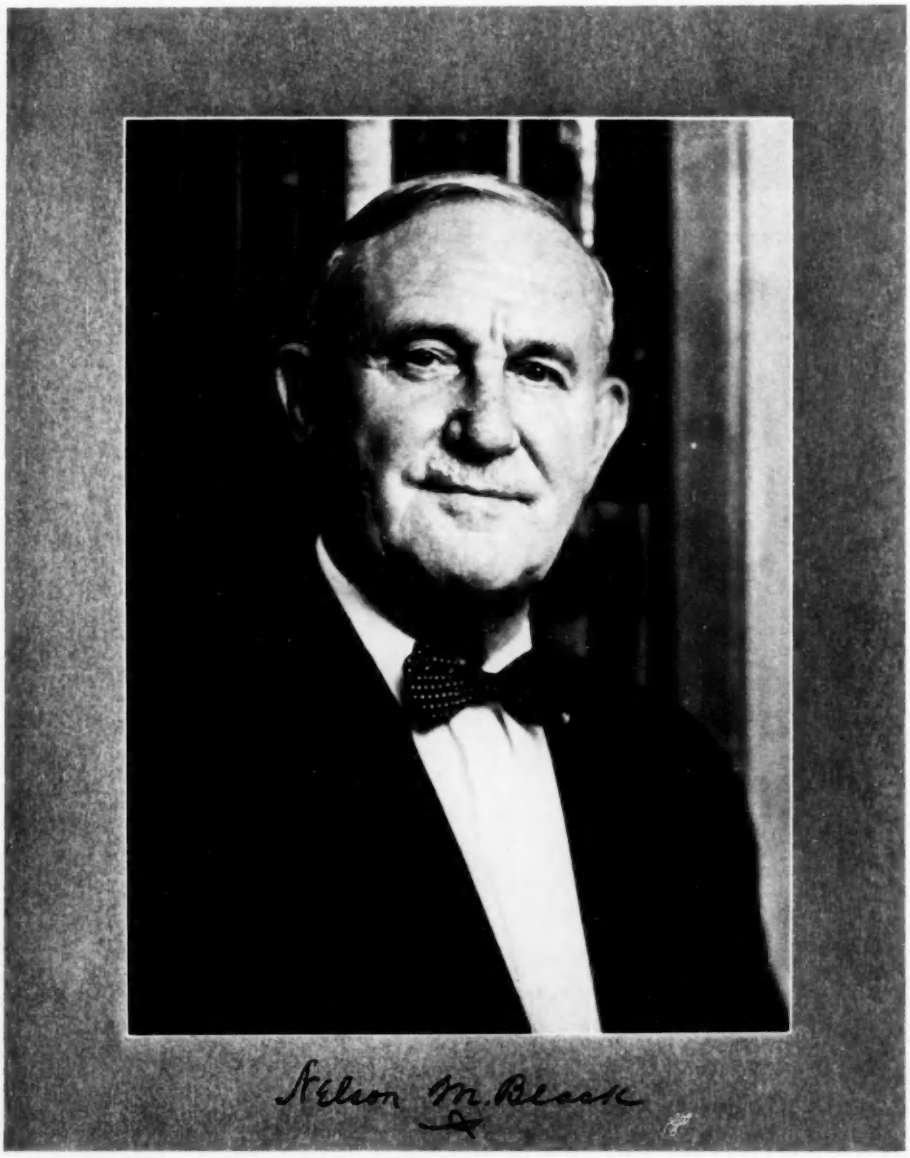

Nelson M. Beach

Nelson Miles Black, M. D.

MIAMI, FLORIDA

Doctor Black was born in Mt. Vernon, Illinois, January 21, 1870. After internship at St. Barnabas Hospital, Minneapolis, he became assistant at Wills Ophthalmic Hospital, Philadelphia. Then, following two years of association with Doctor H. V. Wurdemann, Minneapolis, came a long period of independent practice in that city, 1899-1929, and his removal to his present residence, Miami, Florida, where he is a member of the staff of St. Francis Hospital, and consultant at the Jackson Memorial and Victoria Hospitals.

Doctor Black served in the National Guard and in the Spanish-American War. During the World War, he held the rank of lieutenant-colonel in the Medical Corps, was in charge of the section of ophthalmology in the Surgeon General's Office, and Consultant to the American Expeditionary Forces. He served throughout the war, in this country and at the front. He ranks as colonel in the Medical Corps of the Officers' Reserve Corps.

Among the groups in which Doctor Black holds membership are:

The American College of Surgeons (Fellow).

The American Medical Association (Chairman of the Section of Ophthalmology, 1920-1921).

The American Ophthalmological Society.

The American Academy of Ophthalmology and Otolaryngology (First Vice-President, 1933).

Wisconsin State Medical Society.

Florida State Medical Society.

Doctor Black is a contributor also to various scientific publications, and has been the recipient of many honors.

The Academy is pleased to honor, and appreciates the privilege of dedicating the TRANSACTIONS *of 1939 to Doctor Nelson Miles Black, Miami, Florida.*

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Joint Session

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Joint Session

The American Academy
of Ophthalmology and Otolaryngology

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Forty-Fourth Annual Meeting
*Chicago * 1939*

Address of the President

* * *

TREND IN ACADEMY PROGRAMS

GEORGE M. COATES, M.D.
PHILADELPHIA

FELLOW MEMBERS of the Academy, officers and guests, and our Honor Guest, Doctor Burt R. Shurly. Last spring, in the Bulletin, I promised you that the program presented at this meeting would be unsurpassed, and that the presidential address would be short. In the program in your hands, you will see the confirmation of the first promise — and I am now proceeding to make good the second one. This will be no scientific discourse.

Many of my eminent predecessors in the high office to which you have elevated me, have traced the glorious history of our Academy much better than I can, and while I shall not review this history in detail, I shall make a few remarks upon the recent trend in Academy programs which has influenced graduate training in Ophthalmology and Otolaryngology over the whole country.

The Century dictionary defines "Academy" as a place "where one goes to learn," and this is a true definition of our Academy, for undoubtedly everyone who attends does so with this main object in view. In the twenty years since the original Round Table discussions were instituted, the character of planned instruction has evolved from the general discussion of a given subject under a leader, to highly developed courses of instruction. As now given the popularity of these courses attests to their real value and new teachers are being constantly developed to fill needs for specific types of instruction. Other societies and groups have followed the lead of the Academy in this respect. The training and experience developed in these courses has started many a young man up the ladder of university teaching in graduate, post-graduate and undergraduate work, which has been of inestimable value, not only to the individual but to the organization or institution which he serves. Think back to the early meetings of this Academy when

but a half dozen round table discussions were offered and compare them with the huge instructional program presented at this meeting for which many hundreds of course tickets have been sold. Think also how the seed planted by the Academy has sprouted, grown and born fruit in the crops of post-graduate groups in all sections of the country where this idea has been utilized, and which comprise county and state societies, local societies, sectional groups, the various post-graduate groups, not only in our two specialties but in all the other specialties as well, and even in many national organizations. We should be proud of this pioneer work, and still more proud that we have always progressed and kept ahead of the game by the inclusion of new subjects of timely interest and improvement in the technic of the courses themselves. Perhaps the latest development, that has not yet reached its climax, is the continued or serial course such as is now being given in pathology and in seventeen other subjects. The popularity of these courses of instruction is such as to, undoubtedly, forecast similar extended courses where the time limit of one and one-half hours is not sufficient to give in detail the information the members desire.

At this writing, all of the major specialties in medicine have set up Boards of examination for certification of competency to practice, and here again the Academy was the leader, for out of its own two committees to examine candidates for membership grew the American Board of Ophthalmology, this week celebrating its silver anniversary. A few years later the American Board of Otolaryngology was formed, and for many years, until very recent times, these two were the only ones in the field.

As your President, for the time being and for the course of this meeting, and in behalf of the officers and Council, I desire to extend a most cordial welcome to our guests, those from other countries as well as those from our own. We hope they will feel at home with us and will take part in our discussions and that they will, when eligible later on, join us in membership and help us to carry on the work of the Academy to ever better and greater things.

Those of you who have carefully studied our program for this meeting will have noticed the part that our colleagues of the Army Medical Corps are taking in our work this year. The place of Ophthalmology and Otolaryngology in Aviation was established by members of this Academy in 1917 and the older members will well remember the examinations in these branches conducted in the Army at every large camp and cantonment for would-be pilots and observers. As Doctor Fenton has well said to Surgeon General

Magee, we hope that the time will soon come when these examinations of pilots will again be largely a function of those trained in ophthalmology and otolaryngology and that it will interest many of our younger members in taking up the work of flight surgeons.

This, it seems to me, is a matter of the utmost importance, made necessary by world-wide conditions, in regard to both commercial and military aviation, and by the expanded program for the training of pilots in our high schools, colleges and universities. America cannot have too many qualified pilots for her national defense, or for commercial pursuits if war spares us. I would urge the younger men, therefore, to study this subject with interest and prepare themselves for this work. In the last Great War it was necessary first to train examiners, but the next one, if it comes to us, should find us well prepared in this respect, this time under the guidance of the officers of the Army Medical Corps who have given the subject special study and are now prepared to impart to us the results of their work.

An important and constantly expanding part of the duties of this organization is the work for the handicapped school child. For long years, our ophthalmological members have lead in the campaign for the Prevention of Blindness, as you all well know. Through many discouragements and by arduous work, great results have been obtained and the citizens of this country have benefited immeasurably thereby. A similar work for the Preservation or Conservation of Hearing has unaccountably lagged behind in spite of the efforts of a few devoted otologists who have labored early and late in this cause, and given unsparingly of their time and energy. Undoubtedly, the dramatic appeal of the blinded to our sympathies is lacking in the care of the deafened, but the number of seriously deafened is great—the mental misery and economic loss occasioned is enormous. It is perfectly possible to accomplish tremendous good in this work if sufficient interest can be aroused amongst the laity and the profession alike. We know most of the causes of impaired hearing; we know, in most instances, how to avoid this result, but we do not know how to correct the damage once it has become great. Our efforts, as otologists and members of the Academy, should, therefore, be directed with increased energy to spreading the gospel that deafness can often be prevented and we should work unceasingly to this end, by interesting all physicians, otologists as well as others, in this campaign, and by cooperating wholeheartedly with the local and national organizations of the deafened laity to this end. These latter

have done and are doing a splendid work, under great difficulties, for their deafened members, supported by only a handful of otologists. I urge every one of you to become interested in the cause of the deafened, to cooperate with the local Leagues for the Hard of Hearing and with the American Society for the Hard of Hearing, by giving wise and understanding council, by attending their meetings, by aid in the establishment of local chapters where none exist, by starting deafness prevention clinics, of which there is at present a woeful lack, and by sponsoring legislation for the nation-wide hearing examination of school children. It is obvious that if we hope to combat the end results of progressive deafness in any of its forms, and they are many, these cases must be detected in their incipiency, which is usually in childhood. The detection of impaired hearing in the mass of children of pre-school age is a difficult problem but can be accomplished in time by education, propaganda and the increased establishment of prevention clinics. We know how the problem can be handled in the schools, although improvements in method and technique will doubtless be made. What is needed is increased interest in this matter which will lead to legislation and the provision of funds sufficient eventually to examine yearly, by approved methods, every child in every school in the land. This is a big order, only to be accomplished in time. Dare anyone say it is too big for the united membership of our great Society to bring to a successful conclusion? I do not think so.

And now a word in closing about this office which I now hold and will in a few days relinquish to a most worthy successor. I need not say how deeply I appreciate the honor you have bestowed upon me in electing me your president. No greater honor can come to an American Ophthalmologist or Otolaryngologist. I thank you from the bottom of my heart for this expression of your confidence, for your friendship and friendliness. Perhaps the latter seems to me to be the more important. The Academy is a friendly organization — friendships formed here are numerous and lasting, and friendship is more to be desired, often, than other human contacts. May our Academy, under the guidance of its wise Council, its splendid corps of secretaries and its sound financial leadership, long continue to prosper and be a beacon to students from near and far to come to it as a "place where one comes to learn."

And so, fellow members, the address of the President comes to its conclusion. You will now listen with greater pleasure and profit, I am sure, to that of our Honor Guest, Doctor Burt R. Shurly.

Address of the Guest of Honor

HANDICAPPED CHILDREN IN RELATION TO OTOLARYNGOLOGY

BURT R. SHURLY, M.D.
DETROIT

EDUCATION is undergoing profound changes together with all economic and sociologic phases of modern life. If these changes are constructive and to the benefit of humanity individually and collectively the medical profession and specialists in particular may well apply the constructive ideas and join together in promoting that which is good. We are faced on every side with a destructive war where human life is sacrificed and the living survive with some tremendous handicap. Those that concern our specialties — blindness, defects of speech, facial and jaw deformities, deafness — that are the results of war wounds, demand special surgery and rehabilitation.

These problems were met with skill and efficient surgery in the last World War. Scientific improvement in surgery will be shown in our next World War if this becomes a necessity.

I would speak particularly of those who would not be available for enlistment and service because of congenital or acquired defects such as blindness, loss of hearing, speech and jaw defects. The failure of early diagnosis and the neglect of treatment of the school child is outstanding. The enlistments in the last World War gave us statistical findings that refer to the number of defectives and their rate of increase in each state. The pauperization of people and the use of welfare and dole funds, charity beds, tax moneys and public charges in general have much to do with medical economic problems.

Those of us who serve on Boards of Education know that the cost of medical care is increasing and that when we are called to

care for those who are handicapped our expenses may be lessened and our educational facilities strengthened by the early diagnosis of defective vision and hearing.

Medicine will not be socialized if a card index of every school child shows the name of the family doctor and the name of the parents. They are each sent a report of the findings of examination and a proper reference can be made when the family doctor desires more information. I present a slide of the index card and a report to be sent to the family doctor, as used in the Detroit public schools.

A hearing survey of Detroit school children published in the Bulletin of the American Academy of Ophthalmology and Otolaryngology May, 1939, showed that of 7,959 completed examinations, 392 or 4.09% of the children had impaired hearing. This project was financed entirely by the Board of Education of Detroit and the Detroit Board of Health, Doctor H. F. Vaughan, Health Officer, and under the immediate direction of Doctor Joseph G. Molner. No U. S. Government funds were used except for helpers. This work can be done in any local community with the same cooperation. Information in regard to the details may be obtained by communicating with the Detroit Board of Health or Board of Education.

Tests for forty children at a time with 4A or 4B audiometer is only a screening test and subject to errors and must be repeated once or twice in 29%. The final check and supervision by an otolaryngologist is required as to the mechanical conditions of the audiometer, condition of the testing record, the needle, the child as to attention, nervousness, intelligence, the quiet of the room, as may all be factors in accuracy.

The WPA project is nation-wide and may be established in any state, with the consent of the proper health officer. In Michigan it calls for the testing of 650,000 children enrolled from the third through the twelfth grade in all school systems.

The purpose of the project is to ascertain the hearing conditions of all children and to adopt such measures as will effect the facilities of the pupil for learning and whenever possible avoid unsatisfactory progression of abnormal conditions. The teaching of lip reading may be carried on as a WPA project as done by the New York Board of Education. A brochure may be obtained that describes these activities from the chief of the Planning Section Education and Field Office in New York City.

Illinois has a visual and hearing test project in Chicago under the leadership of our past president, Doctor Harry S. Gradle. The problems of human safety with the ever increasing number of automobile accidents is now the subject of wide study under a special committee of the A.M.A.

The testing of school children for visual and hearing defects and color and night blindness with special restrictions for those unqualified as drivers may have some influence in reducing the frightful loss of life while our streets remain a battlefield. This great society may well foster some standardizations of limits in these defects beyond which the safety of the driver and pedestrian is in danger. We have in some of our high schools a driver course which recommends for license those physically and mentally qualified. Is not this a good place to begin restrictions and eliminate a measure of extra danger.

Visual acuity and muscular balance will be determined by the Snellen Chart and the cover test, color perception by the Ishihara test. Defects will be recorded and submitted to the nurse, health department, parents, and family doctor for obtaining corrective or remedial measures. Local participation in any community will be voluntary and by request.

Resolutions of endorsement, support and encouragement in the testing for defective vision and hearing have been passed by our society, the A.M.A., various state, local and special societies through these many years but it is only recently through Boards of Health and Education and governmental and professional aid that this work is actually going on as a nation wide project.

With great wars under way carrying world wide destruction to life and limb the repair of the handicapped becomes more and more necessary. Wounds of war and congenital and hereditary defects will demand much of our profession. Only the fit can serve in the defense of their country. An examination of the physical defects among our first million men drafted in the World War demonstrated the weakness of the race. A few slides will show these defects of interest to our specialties.

In the Detroit school system with 268,000 children and the private and parochial schools with 80,000 additional the teacher, the nurse, the school physician, the parents and the family doctor form an ever alert group guarding the health and diagnosing any deviation from the normal of the school child. Backward or ab-

normal conditions are referred to the Psychological Clinic where proper classification may be made. In our own state of Michigan, which is in the so-called goiter belt, it was found advisable in Detroit to establish an endocrine clinic under the able management of Doctor Carleton J. Marinus and assistants. Children of goiterous families and those who showed other disturbances which are manifest in eyes, ears and throat, were given endocrine examination with the consent of their parents and doctor and the findings reported to them.

We feed more than 50,000 children a day in the lunch rooms. An opportunity to use iodine-salt is therefore afforded. The two surveys for goiter in lower Michigan in ten years show a remarkable decrease. Pregnant mothers are advised to take iodine and beneficial results to their offspring are assured.

The glandular clinic during ten months cared for 4,201 visits of new and old cases. These were referred to the family physicians whose names could be secured (40% of this group). Of 158 family doctors all but eight were cooperating. The remainder of the group were cared for or referred to some other agency or hospital clinic.

Schools cannot handle those with an intelligence quotient of less than 50 to 59%. While the improvement may be marked under treatment we cannot justify ordinary school education or self dependence. As adults they require special education at much greater expense. Treatment is necessary through a period of at least three years. With an I.Q. of 75% or over many brilliant results are obtainable. Goiter, thyroid deficiency and pituitary disfunction included 90% of those under observation and these are of particular interest to the eye, ear, nose and throat specialist. The importance of this work in every large school system cannot be overestimated.

The medical department of Wayne University is under the jurisdiction of the Detroit Board of Education and has the only municipal medical college in the world. It is therefore possible to correlate the various activities including research under the various departments.

A survey of the epileptic children in the school system showed some 400 in number. Many of these gave disturbance daily in the ordinary classroom. It was therefore thought advisable five years ago to establish an epileptic hospital school where these children could have observation night and day under competent teachers,

nurses and medical men. This became necessary because the state institution had a waiting list of 700 and had no educational facilities whatever. A large, partially vacant school building was equipped to care for 168 of these unfortunates and Doctor O. P. Kimball and a medical staff was appointed. A study and survey was made of this problem including the hospital school cases and those in the out-patient department. From September 15, 1938, to June 15, 1939, 520 cases were studied. During the period 121 children were given Dilantin 1½ grain t.i.d. — an anti-convulsive drug with the formula of sodium diphenol hydontoinate. Dilantin given for three months' treatment controlled seizures in 26⅓% of the cases for a year or more; 44% were improved and 30% showed little or no improvement. They were under the care of a dietitian with carefully regulated and studied diet.

While epilepsy may be remote from our specialty, the use of dilantin developed after three months an interesting and bizarre research in otolaryngology. The toxic effect of the drug showed in 50% of our cases a marked hypertrophy of the gums so that the gums in some cases completely overgrew the teeth. No blood changes could be demonstrated. The phenomena in some respects resembled scurvy and a deficiency of ascorbic acid or Vitamin C was determined. Many observers were puzzled as no one previously had seen this pathology. The condition subsides in two or three months when dilantin is withdrawn, and is modified by excessive doses of Vitamin C but not removed. The pathologic report shows marked hyperplasia of the connective tissue and mucous membrane with loss of intercellular cement tissue. The Clinic under Doctor Kimball's direction has observed more than 1,000 cases, 35% of whom have direct chromosome linkage.

Survey of Detroit School Children

Phase I — Hearing *

Purpose:

To ascertain hearing conditions of all children enrolled in classes from the third through the twelfth grades in all school systems, and to make provisions for the medical care and education of those needing such service because of hearing defects. To save large numbers of children from the handicap of becoming hard of hearing in later life, and the effecting of remedial measures immediately to improve the learning facilities of the pupil, and to avoid the unsatisfactory progression of such abnormal conditions.

Phase II — Vision *

To ascertain vision defects of all children enrolled in classes from the third through the twelfth grades in all school systems, and to make provisions for the medical care and education of those needing such service because of defective vision. To bring about immediate corrective measures to improve learning facilities of the pupil and to avoid unsatisfactory progression of such abnormal conditions.

Description:

Hearing loss of all children will be determined by the use of a 4A audiometer. Those showing a loss of 9 SU or more will be retested by a 4A audiometer in order to eliminate fear, ignorance or learning factors. Of the retested group, those which show 9 SU or more hearing loss will be tested individually with a 6A audiometer and examined by an otolaryngologist. As a result of this last examination findings will be submitted to the teacher, nurse and health department officials, who will in turn notify the parents and family physician of the conditions, and assist in obtaining corrective or remedial measures.

Visual acuity and muscular balance will be determined by the simple Snellen Scale and cover test. Color perception will be determined by the Isohari test. Visual defects will be recorded and submitted to the teacher, nurse and health department officials, who will in turn notify the parent of the condition and assist in obtaining corrective or remedial measures.

Objectives to Be Served by the Project and Justification:

To determine hearing loss of all school children from the third through the twelfth grade in all school systems in the State of Michigan. To obtain data on hearing loss and the condition of hearing organs of those children who show a significant hearing loss. These findings will be relayed to officials concerned and to the parents and their family physician, for corrective and remedial measures.

It has long been recognized by medical authorities and educators that there is an important relationship between a child's educational program and his physical well-being. Likewise, great importance to the future progress and development in adult life is the prevention of unsatisfactory progression of abnormal conditions in the child. Certain physical defects are prone to impede the child's ability to learn, most important among these select defects are disturbances in hearing and vision.

Through the ages, a persistent attempt has been made to restore the hearing of the chronically deafened. To be sure, much can be done for many of these with beginning deafness — if it is done in time. The universal experience of the otolaryngologist, however, seems to be that patients come to

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* The purpose of this project is to test and examine all school children for vision and hearing defects and the project is so drafted that sufficient personnel and materials will be provided to accomplish this purpose. However, this project will be placed in operation only in those communities where there is a specific request for participation. One of the important duties of the State Supervisor of this project will be to interview school and health officials in each county in order to determine whether or not this project is desired by the local authorities. The local participation will, in all cases, be voluntary.

them too late. The well informed otolaryngologist knows that his greatest service to such patients is to face the same situation that has always confronted the Ophthalmologist — to prescribe an artificial aid. In brief the national problem of vision defects and deafness lies in prevention. There is no better way to approach the prevention of blindness or deafness than the systematic and universal examination for hearing and vision of school children.

SCOPE OF THE PROJECT:

All examinations will be made in school buildings. The necessary arrangements for the conduct of this project will be the responsibility of an executive group of supervisors, but in particular that of the whole time state-wide project supervisors. In each of the schools, the principal will be consulted and arrangements made to use a large quiet room, large enough to house 40 children with adequate light and ventilation. A time schedule of examinations for the children in the school will be determined in advance, using a forty-five minute interval for each test of 40 children. Some 650,000 children will be tested for hearing loss. It is estimated that some 130,000 will receive a second 4A audiometer test and some 35,000 will be tested with a 6A audiometer and examined by an otolaryngologist.

SOURCE OF DATA:

All school children enrolled from the third through the twelfth grade in all school systems of the State of Michigan, where expression of desire for participation is obtained from local authorities.

WORKING PROCEDURE:

General — Personnel of Unit and Procedures:

A Physical Scientist will serve as spokesman and supervise the hearing test.

The following is a resume of Direction procedures. (Form H hearing test.)

4A - 4B AUDIOMETER HEARING TEST

When pupils are seated — Is there anyone who does not have a pencil?

Most of the children who have taken this hearing test have enjoyed it and I know that you're going to enjoy it too. I don't want you to feel nervous because it's an easy test to write and I know that you'll do your best.

There are two things that you must remember: first, the room must be very quiet during the test and second, you must listen carefully to the directions and follow them.

This hearing test consists of listening to two records, one for testing your right ear and the other for testing your left ear. You will hear the voices of a woman and a man. Their voices will be loud at first and then become weaker and weaker since the people are moving away from you while talking. They will call numbers which you will write on your papers.

You will notice that there are columns 1, 2, 3 and 4 under the right ear form on your papers and columns 5, 6, 7 and 8 under the left ear form, you will write the numbers you hear in the first column. The numbers will

be called in this way: "5 2 6" (write the number on the board on a similar form). Now write "5 2 6" in the first space in the first column; write "3 4 8" (on the board). Write "3 4 8" in the space below. Put your pencils down.

The numbers in the first part of each column will be easy to hear but as you get to the bottom of each column, it will be more difficult to hear them since the voice is becoming weaker and weaker. If you can't hear the numbers, don't get excited; sit quietly in your seats and wait until you hear the voice on the record tell you when to start writing the numbers in the second column, etc. Do not write the numbers in the next column until you hear the directions on the record. You must write all the numbers you hear. For example, if the number "3 2 4" should be called and you hear only "3 2" or "2 4" or "3 4," write those numbers in the space and go on to the next number. In other words write all the numbers you hear because it will make a difference in scoring your papers.

A very important part of this hearing test is your being able to put on your ear phones properly. Watch and listen carefully to the instructions. (Demonstrate, using an ear phone.) The cord must be straight down, the holes in the receiver must be placed directly over the opening of your ear because the sound is transmitted through these holes into your ear. Pick up your ear phone with both hands and stretch the wire of the head band. Place the ear phone over the ear that is being tested and then bring the wire headband over the top of your head. (Give this direction twice to third and fourth graders and to other groups if necessary.) Be sure your ear phone is on properly so that you'll hear properly. Now point to your right ears. Girls, you must pull your hair behind your ears because it makes a difference in your hearing. Now put your ear phones on your right ears.

Do not write. I'm going to check your ear phones. If you can't hear raise your hand. Now listen to your last minute instructions. Do not write the numbers "5 2 6" and "3 4 8" again; when you hear them on the record, listen and check them on your paper. Write the number you hear after "3 4 8" in the space below. Write all the numbers you hear in the first column and wait until you hear the voice on the record tell you when to start writing in the second column. Sit very quietly and do not shuffle your feet during the test. If your pencil breaks raise your hand quickly. The attendants will help you. Ready. (They write the test for the right ear.)

Now put your pencils down and take off your ear phones. Put them on your desks and stretch your arms for a minute. Put your ear phone on your left ear and be sure that you put it on properly. I'll check your ear phones and if you can't hear raise your hand. Write all the numbers in the fifth column under the left ear form. Ready.

History

Fill in the top of your paper — Your first and last name, age, grade and section and the date. The date is on the board.

There are questions on the bottom of your paper which you must answer, listen carefully to your instructions. The attendants will help you.

The first question — Did you ever have an ache or pain in your ear? If you did write "yes," if not, write "no." The children who wrote "yes"

must fill out the questions "which ear?" and "when?" I have written some words on the board to help you answer the questions. In answering "which ear?" you must tell if it was or is your right, left, or both ears. In answering "when?" you must tell if you are having trouble now, sometimes, often or how old you were or how many years ago or you may remember the year in which you had the trouble (1935 or 1937, etc.).

The second question — Did you ever have a running ear? If the wax in your ear becomes so soft that it continues to run out of your ear or if there is any discharge it is called a running ear. Write "yes" or "no" and the children who write "yes" must tell "which ear" and "when."

The next question — Does your ear run now? Write "yes" or "no."

The last question — Do you ever hear noises in your ears such as ringing, humming, buzzing or roaring? If the only time you hear such noises is when you have a bad cold, your answer should be "no" because we often hear those noises at that time. If you hear the noises frequently or almost all the time, write "yes" to the question and tell "which ear" and "when."

Are there any children who have had a mastoid operation? Write a sentence on the bottom of your paper telling "which ear" and "when."

Have any of you ever had your ears lanced? If so, write a sentence and tell "which ear" and "when."

Have any of you ever had an abscess in your ear? Write a sentence on the bottom of your paper telling "which ear" and "when."

Sometimes when we have colds they affect our hearing. If you have a bad cold or a slight cold will you write across the top of your paper — "I have a bad cold" or "I have a slight cold."

The attendants will pick up your papers now. Take your pencils with you and watch the cords in the aisles and be careful not to pull the ear phones off the desks.

Two physical scientists aides will be on hand to assist the pupils in properly adjusting the ear phones, answer pupils' queries and adjust difficulties. Observe the progress of the test and render whatever assistance is necessary.

Particular assistance will be necessary when the history part of the test is being executed. At the completion of the test the papers will be checked before the pupil leaves and collected. Each group of tests must be kept separately and marked to indicate grade level, date and time of test and serial number of machine used. The ear phones are then cleaned and room placed in order for the next group to be tested. The new group is called for and seated ready to take their hearing test.

Scoring Crew: 4 Junior Clerks

Two clerks will serve as graders of the preceding group tested. The time required to grade and score each test paper is approximately 3 minutes. One clerk will serve to recheck the scoring, one clerk will sort the group scored into those showing 6 S.U. or less hearing loss (normal group) and those showing a rating of S.U. or over (group with defective hearing). The papers found in the normal group will immediately be referred to and the findings transcribed onto the pupil's health record card which is available

in each school (Exhibit I). The remaining group showing defects will be kept intact and placed in a paper folder, the normal group will likewise be kept intact and submitted to the central office.

Procedure in Central Office

On receipt of the scored hearing forms records will be made on date, place, school grade and per cent of children with normal hearing by sex and those with significant hearing loss. The group which shows a significant hearing loss will be returned to the principal's office of the school where arrangement will be made to retest the defective group in the same manner with a 4A or 4B Audiometer. Procedures in the field will be identical to the first test.

For those in the normal group. Form II (Parents Report) will be made for each child and submitted to the principal's office, where the nurse and school teacher will be responsible for the relaying of this information to the parent.

On receipt of the second scored hearing forms: records again will be made on the original forms kept for that specific group in the central office. Those showing 9 S.U. or more hearing loss, will have the pertinent information recorded and again submitted to the school principal's office. There to be held until arrangement is made for the final test to be made by the otolaryngologist and his staff.

The findings of the final test when returned to the central office will be noted and final tabulation of the particular school prepared. The findings of the otolaryngologist will be noted on Form III (Parents Report) and sent to the school principal's office, where the nurse and teacher will be responsible for the relaying of this information to the parent and further attend to the follow-up work which will be necessary for the child's welfare.

Vibratory or Frequency Test

The group of children who have revealed a hearing loss of 9 S.U. or more on the second test will have appointments made by one of the clerks working with the otolaryngologist, to have physical examination of the hearing organs, in addition a frequency test by one of the attendants. No more than three tests and examinations per hour will be possible for the otolaryngologist and his attendants. The clerks will have to grade and check the final frequency test and note the physical findings of the physicians on the reverse side of the Form H (Second hearing test). The cumulative findings are then transcribed onto the pupil's health record card which is available in each school (Exhibit I). The Form H completed is then mailed into the central office for final tabulation.

When Phases of Work Call for Data Collection

1. *Method*

   a. *From Existing Sources*

   Not applicable.

b. *Collection Forms*

Form H. — Hearing Test.

Form II. — Notice to Parent.

Form III. — Notice to Parent and Physician's Report.

Exhibit I. — Health Record.

2. *Working Instructions* FOR FILLING DATA COLLECTION FORMS
Workers' instructions are given and discussed in IV-A.

WHEN PHASES OF WORK CALL FOR TABULATION OR SUMMARIZATION OF DATA

1. *Method*
Hand tabulation will be used and percentages determined by use of calculators.
c. Tabulation (continued)

2. *Summarization* FORMS OR TABLES
Form VI through XXIII — Audiometer Study
(See pages following)

3. *Instructions* FOR MAKING TABULATIONS AND SUMMARIES
Instructions for tabulation will be determined by the State Supervisor when specific need and type of information is determined by the advisory group.

RESULTS DESIRED

A. *Publication*

It is the aim of the technical advisory groups to compile a report of findings of hearing loss and vision defects discovered in the school population. These reports will be made available to all agencies and individuals interested in these data.

B. *Availability of Collected Data and Reports*

As described in the General Working Procedure, reports and findings will be submitted to school authorities, health departments, parents and family physicians continually throughout the entire duration of the project. The final reports will be accessible to any authorized individual for review in possession of school boards and health departments.

NOTICE TO PARENT

Date.....

Under the Department of Public Instruction sponsorship, a hearing test was given your child BETTY JONES. We wish to inform you that at the present time the result of the test was satisfactory. This means that your child's hearing is normal within the limits of this test. Many children do have hearing difficulties and we are pleased to give you this satisfactory report.

DIVISION OF SCHOOL HEALTH SERVICE

FORM II

Supplementary Information

PHASE II — VISION

I. *Objectives to Be Served by the Project and Justification:*

To determine defects in vision of all school children from the third through the twelfth grades, in all school systems in the State of Michigan. To obtain data on vision acuity, muscular balance, and color perception. These findings will be relayed to officials concerned, and to the parents for corrective or remedial treatment.

(For further Justification and Objectives see Item 27, Phase I)

II. *Scope of the Project:*

All examinations will be made in school buildings. The necessary arrangements for the conduct of the project will be the responsibility of the executive group of supervisors, but in particular, that of the whole-time project director in charge. In each of the schools where examinations are to be held, the principal will be consulted, and arrangements made in advance to use a large, quiet room with adequate light and ventilation. The time schedule of examinations for the children in a given school will be determined in advance. It is estimated that the time necessary for a complete visual test will require approximately twenty minutes per child. In addition, intensities of light in selected schools or upon request will be made with a simple light indicator to facilitate seating arrangements for those children showing defective vision. This phase of the project will also facilitate programs during school vacation periods. It is anticipated that children in orphanages and institutions of similar description will likely have their vision and hearing tested during the summer months.

III. *Source of Data:*

All school children enrolled from the 3rd through the twelfth grade in all school systems of the State of Michigan, when expression of desire for participation is obtained from local authorities.

IV. *Working Procedure:*

A. *General*

PROCEDURES AND PERSONNEL

A physical scientist will serve as head of each vision testing unit and will, in general, supervise the conduct of the three phases of this test. The physical scientist will give a visual acuity test with the simple Snellen Chart and will determine rapidly the muscular balance by a simple covering of one eye with a card. Color perception will be determined by use of the Isohari test.

An assistant clerk will make notations on Form E. Two other assistant clerks will be used to grade the Form E, and divide into two groups — the group which shows normal vision in all three tests, and the group which reveals some defects in vision. Transcription of findings from Form E, when checked and graded, will be made on the pupil's school health record card. All of the Forms E will then be submitted to the central office for final tabulation.

INSTRUCTIONS TO EXAMINER

The name, age, sex, school and grade of the child should be filled in on the visual test form. The date of the examination should also be noted. For the testing of visual acuity we shall use the Snellen Chart, or modified Snellen Chart frequently called the "E" Chart.

Persons wearing glasses should have their eyes tested with the glasses and again without glasses. The visual acuity under both conditions should be noted. In doing the test, the test chart should be placed at a distance of 20 feet from the patient and at a level roughly comparable with the level of his eye. The chart should be well lighted, preferably by natural light rather than artificial.

In doing the test, the patient should stand at the required 20 feet distance with his back towards the windows, if the windows are the source of illumination. Cover the eye opposite the one that is to be tested, with a dark piece of cardboard and make sure that the patient is directly facing the test chart. Ask him to read the letters on the bottom line. If he successfully reads these letters, proceed to the line above. These lines, as you will note, are marked with the figures 20, 30, 40, etc. In recording the visual acuity of the patient, the numerator remains constant and the denominator denotes the line on which he failed. For instance, a visual acuity of 20/40 indicated that at a distance of 20 feet the patient could not satisfactorily read the 30 line.

Usually, it is best to start testing the right eye and after having completed the test of the right eye, proceed to the left eye, recording the visual acuity for each eye.

The muscle balance test can be performed at the same time that the visual acuity is being taken. Muscle balance is definitely known to have some bearing upon visual acuity. Muscle balance is noted as negative or positive. By this we simply mean that the eye which has been covered and has been out of function during the test of the opposite eye, remains in focus with the eye that is being tested, or else that it has been out of focus with the functioning eye, and has either turned inward or outward. If the eye has turned inward or outward, the test is considered positive. This can be noted by quickly removing the cover from the eye and noting whether the eye which has been covered turns inward or outward after the cover is removed. As has been stated before, if the eye remains fixed, the test is negative. Note which eye is the fixing eye, and record this on the visual test form. If the test is positive, that is, the eye shows inward or outward motion after being uncovered, record this on the visual testing form. If the eye moves outward or temporally, it is called converging. If, when uncovered, the eye moves inward or nasally, it is called diverging imbalance.

Color perception tests will be performed by the Isohari Test. If the persons recognize the figures on the several charts they will be called negative. If, however, they fail to recognize the figures, in part or in whole, the color perception test will be considered positive. For example, on one of the charts, normal persons will recognize the figures 2 and 6. Those persons who are red blind will recognize only the figure 6. Those who are green blind will recognize only the figure 2. This should then be recorded on the visual test form. These persons are considered partially color blind.

In rare instances, some persons will be totally color blind. Considering a person totally color blind should be guarded against in so much as this is a rare condition and visual acuity may have an important bearing on a person's inability to read the color perception test.

The history form noted on the bottom of the visual test form should be thoroughly filled out before the test can be considered as complete.

Visual Testing Guide

The information on the first three lines must first be recorded.

The chart which you see on the wall is one which we use for the testing of eyesight. Your ability to recognize the black letters and the direction in which the bars point, is the indication of your ability to see well. Read from the bottom line upward and indicate the direction in which the bars of the letters point. Please look at the letters carefully, speak freely, and follow the pointer.

Examiner:

1. Score patient's vision with and without glasses if he wears glasses.
2. Refer to instructions. Note muscle balance, if either eye converges or diverges during the visual acuity testing, record muscle balance test as positive. Record whether the motion is divergence or convergence, that is, does the eye turn inward or outward.

This second test is one which we use for testing your ability to recognize colors. There are a few people who are color-blind. These persons will not read the test charts with the same accuracy as normal persons. Among these general mixtures of dots of various colors, you will recognize a large-sized figure. As for example, a figure 2 or figure 8, covering almost two-thirds of the dotted area, you need not spend a great deal of time in taking the test. If you can recognize the figures you will recognize them almost momentarily. Please read aloud the numbers which you see on the successive charts.

Procedure in Central Office

On receipt of the scored vision forms, records will be made on the date, place, school grade, and per cent of children with normal vision, by sex, and those with vision defects, sub-divided into slight defects, moderate and marked. For all those children who show normal vision, Form IV (Parents Report) will be made for each child and submitted to the principal's office, where the nurse and school teacher will be responsbile for the relaying of this information to the parent.

For those children showing vision defects, Form V (Parents Report) will be made, stating the impression gathered as a result of the test. These forms will be submitted to the principal's office, where the nurse and school teacher will be responsible for the relay of this information to the parents, and whatever follow-up work will be deemed necessary by either the Board of Health, or the School Board.

A. When Phases of Work Call for Data Collection:

1. *Method*

a. *From existing sources*
Not applicable.

b. *Collection forms*
Form S — Snellen Scale Chart
Form E — Vision Test Schedule
Form IV — Notice to Parent
Form V — Notice to parent and physician
Exhibit — Health Record Card **(See Phase I)**

B. Working Instructions for Filling Data Collection Forms:

2. *Workers' instructions are given and discussed in IV-A, Phase II.*

C. When Phases of Work Call for Tabulation or Summarization of Data:

1. *Method*
Hand tabulation will be used and percentages determined by use of calculators.

2. *Summarization forms or tables:*
Tables 34 through 51. (See attached samples.)

3. *Instructions for making tabulations and summaries:*
Instructions for tabulation will be determined by the State Supervisor when specific need and type of information is determined by the advisory group.

V. Results Desired:

A. *Publication*
See item discussed in Phase I.

B. *Availability of Collected Data and Reports:*
See item discussed on Phase I.

Form E

Visual Tests

Name..... School.....
Age..... Grade .....
Sex..... Date.....

Visual Acuity:

	Without Glasses	With Glasses
Rt. Eye	20/	20/
Lt. Eye	20/	20/

Muscle Balance (cover test):
Negative.....
Positive.....

Fixing Eye	Rt.....	Lt.....
Converging	Rt.....	Lt.....
Diverging	Rt.....	Lt.....

Color Perception (Isohari Test):

Negative.....

Positive.....

1. Partial Color Blind . . . .
   Red . . . .
   Red-Green . . . .
   Green . . . .
2. Complete Color Blind . . . .

(over)

NOTICE TO PARENTS

Date.....

Under the Department of Public Instruction sponsorship, a vision test was given your child BETTY JONES. We wish to inform you that at the present time, the results of the test are satisfactory. This means that your child's vision is considered normal within the limits of this test. Many children do have vision difficulties and we are pleased to give you this satisfactory report.

DIVISION OF SCHOOL HEALTH SERVICE

FORM IV

NOTICE TO PARENTS

Date.....

Under the Department of Public Instruction sponsorship, a vision test was given your child BETTY JONES. We wish to inform you that the results of the test were unsatisfactory, and that your child should receive medical attention. Early correction of the underlying cause of your child's defective vision is very important.

DIVISION OF SCHOOL HEALTH SERVICE

(over)

FORM IV

Findings as Result of Test:

.....

.....

.....

.....

.....

VISION STUDY

Age and Sex	VISUAL ACUITY FINDING (Slight)	(Moderate)	(Marked)

Tabulated by schools, cities, counties, state, urban and rural areas.

Tables 34-39

VISION STUDY

Age and Sex	Muscular Balance

Tabulated by schools, cities, counties, state, urban and rural areas.

Tables 40-45

VISION STUDY

Age and Sex	Color Perception
	Tabulated by schools, cities, counties, state, urban and rural areas.

Tables 46-51

DUTIES OF PERSONNEL IN THE CENTRAL OFFICE OF STATEWIDE HEARING AND VISION PROJECT

Administrative:

1. Keeping of time records.
2. Correspondence.
3. General office work and supervision.

Operating and Tabulating:

1. Receipt of daily records.
2. Checking, routing of filing.
3. Typing of Parents' Notice Forms.
4. Mailing.
5. Maintenance of progress reports.
6. Recording of data.
7. Tabulations by schools and areas.
8. Per cent computations.
9. Correlations between defects and their severity according to age and sex.
10. Compilation and reports to be prepared for dissemination to school and health authorities.

Symposium on Essential Hypertension

ESSENTIAL HYPERTENSION FROM THE STANDPOINT OF OTOLARYNGOLOGY

A. C. Furstenberg, M.D.
and
J. H. Maxwell, M.D., *and* G. H. Richardson, M.D.
BY INVITATION
ANN ARBOR, MICHIGAN

In entering into a discussion of this subject, no attempt is made to engage in the acute controversies of recent experimental studies in essential hypertension. Theories and hypothetical considerations are left to the students of research. The text which has been suggested by men in the practice of otolaryngology aims to offer a few personal observations with the hope that they may furnish some practical aid in the diagnosis and treatment of the otolaryngological manifestations of this disease.

Because of the particular interest of our department of Neurosurgery, in the effect of splanchnicectomy upon essential hypertension an exhaustive study of this condition has been made in cooperation with nearly all departments in the Medical School. A trenchant analysis of the records of hypertensive patients has been compiled and all information coded in its minutia and offered by our tabulating system to those of us who may be interested in any of the regional symptoms of hypertensive disease. From this mass of material, I shall refer to a series of 156 cases and discuss the problems presented by this group which pertain specifically to the field of our specialty.

First, we might ask, what is essential hypertension and what etiological factors are responsible for its occurrence? Any attempt to answer these questions is futile because we are still in the dark as to the causal agents or influences in this disease. We recognize

a distinct hereditary tendency because of the numerous reports of cardio-vascular symptoms or accidents which appear in the family histories. The condition is usually discovered after the age of thirty and affects females more frequently than males.

While it is generally agreed by investigators of today that high blood pressure is due to a reduction in the caliber of precapillary blood vessels, the precise cause of these alterations has not been ascertained. "Pressor" substances which are capable of constricting blood vessels have been found in the blood, neurogenic influences have been considered responsible for hypertension and a host of concomitant pathological changes have been observed in nearly all the organs of the body, each proposed at some time or other as the causative agent but none permanently identified with the etiology of this disorder.

From a practical viewpoint, it is of utmost importance that the otolaryngologist recognize the occurrence of varying degrees of hypertension and that in the early stages of the disease, high blood pressure is not a constant finding. One is often surprised to discover a pressure of 180 over 110 today and chagrined when word from the internist to whom the patient was referred, informs him subsequently that the pressure was found to be entirely normal. By the same token it is equally embarrassing to miss the correct diagnosis because one accepted unequivocally, the first reading, which proved to be normal. The fact of the matter is that in the early stages of essential hypertension, a fluctuating blood pressure is a common finding and the examiner should not fail to take several determinations at varying intervals before a final diagnosis is recorded.

The effect of worry and fatigue in causing a rise in blood pressure in hypertensive patients has long been recognized. After a day of vigorous physical effort or a period of severe emotional strain, a blood pressure of 200 mm. H.G. may be noted. Then comes a night of rest and a morning reading within the limits of normal. Too much emphasis cannot be placed upon the evanescent character of the blood pressure in the early stages of hypertension, and the importance of repeated examinations under different environmental conditions, if one is to avoid errors in the early recognition of this disease.

It is at this time that the patient frequently consults the otolaryngologist on account of headache. This symptom dominated the clinical picture in 50 per cent of our cases. Many of these

patients came to us with a diagnosis of sinus disease but in only 12 per cent was the pain frontal in character and of the pressure type that might be confused with that of sinusitis. The headache was frequently nocturnal, dull, boring in character, reaching its maximum intensity in the early morning hours and vanishing completely in the afternoon. Invariably the patient stated that the headaches were aggravated by emotional excitement, nervousness and fatigue.

Another common symptom of essential hypertension occurring in 30 per cent of our patients and next to headache, the most annoying disturbance of the disease, was vertigo. It was usually described as a feeling of unsteadiness or a "swimming of the head" often initiated by a sudden change of position or a quick motion of the head and ordinarily relieved by a short interval of rest. This disturbance was rarely manifested by the feeling that stationary objects were moving in various directions, nor did the patient sense that he was having any great difficulty in maintaining an erect posture. In this respect, the vertigo of hypertension did not simulate that of Meniere's symptom complex. Whereas the latter frequently occurs as a seizure of comparatively short duration and is characterized particularly by violent vertigo which causes the patient to drop in his tracks or seek a recumbent position, the dizziness of hypertension is milder, more or less constant for periods of several hours and not accompanied by those symptoms which indicate some intense stimulation of the equilibratory mechanism. From personal observations and a perusal of this series of cases, I am inclined to pose the somewhat dogmatic statement that a severe attack of vertigo which throws the individual completely out of balance, is in all probability, the classical feature of a Meniere's symptom complex. I do not recall ever seeing a dizzy attack of great violence which proved to be the effect of a neuroma of the auditory nerve or essential hypertension. The vertigo of the latter is more a feeling of instability, giddiness or unsteadiness and these qualities appear to be exceedingly important differential characteristics of the above disorders. In other words, the patient who falls to the floor on account of a precipitous attack of vertigo is in all probability not suffering from an essential hypertension or an acoustic nerve tumor.

Continuing our analysis of the 156 cases of essential hypertension, we find that nearly all of these patients presented emotional disturbances characterized by an inclination to worry, cycles

of despondency or periods of irritability, anxiety and apprehension. One seldom fails to observe in these patients who are physically ill, that they are also psychologically sick and that their fears, worries and emotional disturbances frequently dominate the clinical picture and tax the physician's diagnostic acumen and his patience to determine what symptoms are due to personality conflicts on the one hand, and what are the result of pathological changes of an organic nature on the other. It remains a fact that a large proportion of these patients present a variety of complaints to which we loosely apply the term functional and that when the blood pressure is reduced, as is often accomplished in the fluctuating stage of essential hypertension by appropriate therapeutic measures, these subjective symptoms promptly disappear.

One of the most annoying and occasionally terrifying experiences which these patients suffer is that of hemorrhage. The bleeding usually occurs from the nose and comes from the septal branch of the spheno-palatine artery where it anastamoses with a branch of the descending palatine artery on the anterior portion of the quadra-angular cartilage. Epistaxis occurred in 17 per cent of our series of cases. In most instances the hemorrhage came from the region above described, a most accessible part of the nose and was controlled without difficulty or ceased spontaneously. A number of patients gave vivid descriptions of harrowing experiences with epistaxis but in none of the cases was there evidence of grave loss of blood at the time of admission to the hospital. Nor did we experience any serious difficulty in controlling the bleeding by the usual simple measures of packing the nose with sponges containing adrenalin and the use of a silver nitrate bead or the electric cautery.

It is an astonishing fact that patients with hypertension usually tolerate surgical interference admirably well. There can be no doubt, however, that such persons offer operative risks which are not encountered in individuals with normal blood pressures. Nevertheless it is strikingly true that hypertensive patients ordinarily withstand surgical insults of major proportions without alarming hemorrhage or other vascular accidents. I do not wish to minimize the dangers of post-operative hemorrhage in these patients nor do I desire to leave the impresion that one does not need to be prepared adequately to meet such an emergency. I do wish to say, however, that essential hypertension, particularly the fluctuating stage (malignant hypertension excluded) is not such an ominous

finding and that the apprehension frequently expressed by surgeons when operating on these patients is seldom justified. Care should be taken, however, to avoid unnecessary traumatization of tissues and careful attention must be given to the ligation of all blood vessels of large enough proportions to favor hemorrhage when the blood pressure tends to rise after the immediate shock of depressing operations.

The judicious selection of the anaesthetic tends to guard against severe hemorrhage and other accidents during and after the operation. It is well to hospitalize the patient for two or three days previous to the operation, so that one may determine the character and degree of the hypertension and obtain the benefits derived from rest. If the blood pressure is continuously high and particularly in the malignant form of hypertension, a local anaesthetic devoid of any vaso-pressor agent is the one of choice. Epinephrine should not be combined with local anaesthetics, because of its pressor action and this is expressly true when the hypertension is associated with organic heart disease. If a vaso constricting agent is required, and the advantages of such in nose and throat operations are well known to all of us, cobefrin hydrochloride may be mixed with the local anaesthetic. This is a synthetic preparation related chemically to epinephrine, with the same percentage composition, but with a different structural arrangement. The pharmacological action of this preparation is similar to that of epinephrine but its vaso constricting influence is not so pronounced and its systemic toxicity is likewise believed to be proportionately less. Cobefrin acts for a longer period of time than does epinephrine and it appears to be singularly practicable in hypertensive patients because it does not tend to cause any appreciable increase in blood pressure.

If a general anaesthetic is required, ether, perhaps, administered by the open or intratracheal technique is the safest of them all. Avertin is not a very popular anaesthetic for nose and throat operations because of its depressing action upon the respiratory reflexes and for the reason that patients frequently remain deeply narcotized for long periods of time after the operation has been completed. The dangers of post-operative hemorrhage from the nose or throat of patients who are exposed to the prolonged effects of an anaesthesia are too obvious to mention.

Cyclopropane, the most recent of the inhalant anaesthetics, is of great value in the presence of pulmonary disease, liver damage,

the anemias, blood dyscrasias and diabetes, but in the hypertensive patients it may cause an initial rise in blood pressure and favor hemorrhage or add significantly to the dangers of some vascular accident, such as cerebral hemorrhage. When the blood pressure rises from the effect of this anaesthetic it usually requires a period of 30 to 40 minutes after the induction, before it returns to normal.

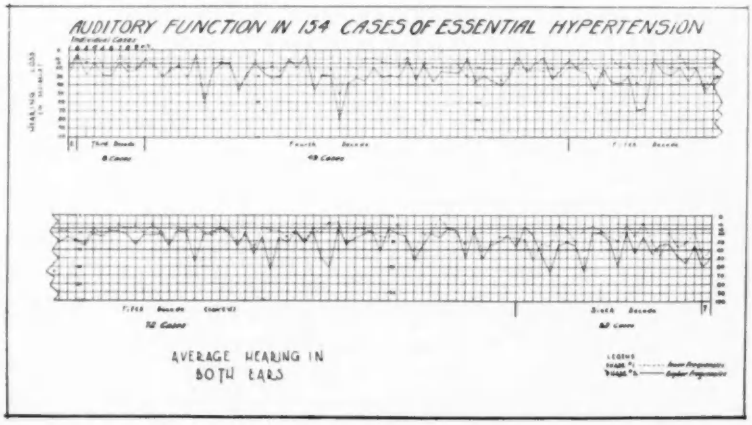
But the point that I wish to stress in connection with this discussion of anesthetics in hypertensive patients is that it is very important that the blood pressure be maintained at approximately the normal preoperative level for the duration of the operation. It must be remembered that the blood pressure in hypertension can go out of control on the slightest provocation and drop precipitously as the result of some depressing agent or surgical shock. When this happens, the heart may suddenly suffer a strain which it cannot endure and the life of the patient is lost. It is an interesting paradox that in order to maintain the blood pressure at a constant level, it occasionally becomes necessary to employ intravenous fluids and some vaso-pressor agent to increase the pressure that is showing a tendency to fall while the operation is in progress. The most useful drug of this character is neosynephrine hydrochloride (levo alpha-hydroxy-beta-methyl-amino-III hydroxy-ethyl benzine hydrochloride) administered in doses of ½ cc. (5 mg.) either intramuscularly or subcutaneously. This preparation requires ten to fifteen minutes for its maximum effect, its influence is felt for approximately 45 minutes and it may be repeated as often as indicated without danger from its immediate effects or from cumulative action. Neosynephrine hydrochloride will frequently support the blood pressure adequately when epinephrine or ephedrine have failed to produce the desired result.

Come now to the last important symptom of hypertension, one which is found in nearly all hypertensive patients and one about which they themselves are seldom concerned, namely, deafness. This feature in the clinical picture of hypertension was the first to attract our attention and excite our interest because of the therapeutic possibilities anticipated from the operation splanchnicectomy. In studying the auditory function in this disease we adopted two principles of procedure. First, it was deemed necessary to establish some standard of normal hearing which would allow for a safe margin of error in the interpretation of the audiogram. Second, it seemed practicable to condense the audiogram in a manner that would permit of a simple graphic comparison of hearing in

the upper and lower tonal ranges. In regard to the first principle, hearing was considered abnormally decreased only in those cases in which there was an average loss of more than 15 decibels. In other words it was concluded that if we would allow a loss of 15 decibels for those frequencies under consideration, we would be making a liberal allowance for all errors that might creep into our particular standard of normal auditory function. In regard to our second principle, the audiogram was arbitrarily divided into two phases. Phase I represented the average decibel loss in hearing for the frequencies of 64, 128, 256, and 512 double vibrations per second. Phase II represented the average loss in hearing in decibels for the frequencies of 1024, 2048, 4096, and 8192, double vibrations per second. We recognized the liberties we were taking with convention in adopting these methods of presenting this problem, but they appeared to be the only means of simplifying our conclusions and expressing them in terms which would eliminate the minutia entailed in a statistical study of this character.

Audiometric determinations were made upon 156 hypertensive patients, of which 154 were diagnosed essential hypertension, while 2 were said to have malignant hypertension. Of this group, 148 members or 96.1 per cent demonstrated significant hearing loss according to the above prescribed standards. The deafness, which was bilateral in nearly all cases, manifested a striking parallelism in the amount of hearing loss and the tone frequencies involved in both ears.

The degree of deafness in this group of patients is represented graphically in the following chart:



Chart

In this graph the loss in decibels represents average for the two ears. The dotted line indicates the hearing in phase I, or the lower tones, and the solid line indicates the hearing in phase II, or the higher tones. As the grouping is by decades it may be noted that most of these patients are in the fourth and fifth decades of life, a period in which one does not expect to find such a uniform degree of deafness in health. The generalization that may be made from this series of cases is that the deafness encountered was in both the low and high tone frequencies but almost constantly more profound in the upper range of the audiogram. This of course, would be anticipated, assuming that the deafness associated with hypertension is of the pure preceptive type. The drum membranes were normal except in a few cases of chronic suppurative otitis media, hearing by bone conduction decreased and the Rinne positive in all of the series. The majority of these patients had not noticed any deafness, and only one-third of them complained of tinnitus.

From this series of cases it may be concluded that essential hypertension is prone to cause some degree of perceptive deafness in nearly all afflicted patients. Insofar as we could determine there appeared to be no correlation between the amount of hearing loss and the degree of hypertension. Individuals with excessively high blood pressures often demonstrated a moderate degree of deafness while patients with hypertension of a mild character frequently evidenced a profound loss of hearing. By the same token there appeared to be no parallelism between the duration of the hypertension and the degree of deafness. Patients who gave a history of a high blood pressure for 20 or more years, often demonstrated a mild hearing loss, while individuals who presented reliable data indicating that they were quite properly classified as hypertensive for only a period of one or two years, often revealed an extreme degree of deafness. Every effort to establish correlations (of this character) or to show parallelisms between the degree of hypertension, the duration of hypertension, and the amount of hearing loss brought forth a confused mass of statistics and abstruse findings which only added to the vagaries of this disease.

An opportunity for further study of deafness in hypertension was afforded in 37 patients of this series who submitted to splanchnicectomy as a surgical means of reducing the blood pressure. Audiograms were made three months following the opera-

tion and compared with the preoperative charts. It was interesting to note that in most patients there was a striking decrease in the hypertension after the operation and that this was associated with some improvement in the hearing. This gain in auditory acuity affected (Phase II, namely) the high tones, but was not observed in (Phase I, that is) the lower tone range. Again it was impossible to demonstrate any correlation or parallelism between the degree of improvement in the auditory function and the amount of reduction in the blood pressure after section of the splanchnic nerves.

Diagnosis

The best way to diagnose hypertension is to keep the possibility of this condition constantly in mind. The symptoms, headache, dizziness, tinnitus and nervous irritability aggravated by fatigue and emotional strain, should lead to the suspicion of this disease and the diagnosis will ultimately be revealed by a series of sphygmomanometer determinations. Again I wish to emphasize that the physician cannot base a positive diagnosis of the presence or absence of hypertension on one (sphygmomanometric) reading. Reference has already been made to the fluctuating character of the hypertension in the early stages of the disease and one must take several determinations at varying intervals before concluding that the patient's blood pressure is within the range of normal.

Another source of error in diagnosis and one for which we should be constantly on the watch, is that committed by the so-called ausculatory gap. The cause of this phenomenon has never been satisfactorily explained although it is encounterd from time to time and is responsible more often than we suspect for a gross error in the diagnosis of essential hypertension. In an occasional patient one finds on inflating the cuff of the sphygmomanometer that the sounds which record systolic pressure disappear at 125, let us say, but reappear at 180. In other words there is a gap of silence between 125 and 180 which is not recognized if one by chance, accepts as final the reading of 125 when the sound first disappeared. In such patients the true systolic pressure will be found somewhere above 180, but it is a simple matter to miss the correct determination if one concludes that the level at which the sounds first disappear is an accurate measure of the maximum systolic pressure. This error, however, can be avoided if one routinely inflates the cuff to 200 or more and allows the pressure to decrease slowly until the first sounds are heard, or better still by

palpation of the radial artery while the pressure in the cuff is being raised and lowered. One must not err by failure to appreciate the significance of the ausculatory gap or by ignoring the simple rules for its detection.

Treatment

It is hardly within the scope of this paper to dwell upon the treatment of hypertension although it seems proper that some mention should be made of the therapeutic measures which deal with the otolaryngological aspects of this disease. An appalling number of drugs has been offered the hypertensive patient; their worth estimated from clinical experience alone, but most of them entirely devoid of any scientific proof of therapeutic value. Nevertheless there are certain fundamental principles of treatment upon which I wish to comment briefly. The patient who is overweight should be placed on a reduction diet and reduced to a level which is normal for him in order to dispose of any circulatory burden upon the cardiac muscles. One must urge the patient to avoid fatigue and undue emotional strain although it is seldom necessary to demand that he give up his useful vocation in life. He may be required to dispense with the innumerable civic and community responsibilities which are frequently thrust upon the willing and obedient citizen. He will do well to give up all strenuous avocations and the many social obligations which are the mark of modern social hysteria, but it is usually for the patient's best interests to permit him to continue with moderate effort, the occupation which provides for his livelihood. Few hypertensive patients are good loafers. They are active men and women animated by a passion for work, energetic in everything they undertake to accomplish and when placed at complete rest, pass rapidly into a phase of mental and physical deterioration.

Careful attention should be given to the avoidance of emotional excitement and worry. One of the evils which besets the life of the hypertensive patient is the frequent visit to the doctor who gravely measures the blood pressure, shakes his head and omnisciently forecasts a dismal future. The sphygmomanometer bears the same relation to the hypertensive patient as the clinical thermometer does to the child of an apprehensive mother. One can abuse the privilege of these important aids in diagnosis to the end that the patient suffers the usual tortures of over-zealous attention and the doctor adds to the burden of his responsibilities in the management of this affliction. It is not necessary to subjugate the patient for the

purpose of frequent blood pressure determinations and serious conferences on the prognostic value of each. Nothing is more detrimental to his peace of mind and likewise his hypertension than the experience of passing through cycles of hope and discouragement provoked by finding the blood pressure low today and high tomorrow. One will do well to treat the affliction as lightly as possible, avoid engaging discussions of the pressure variations from time to time and give the patient every assurance that careful attention to his diet and health habits will prolong his life for many years to come.

It is true that a great many patients who have given rigid adherence to these simple rules of living have remained in the fluctuating stage of hypertension for 25 years or longer. Others, and particularly those of careless habits, have passed rapidly into the so-called fixed stage of essential hypertension in which the blood pressure is continuously high and the annoying symptoms constantly present. In this stage, the period of deterioration may be rapid, terminating in a cardiac "breakdown" or a cerebral accident.

On the theory that resection of the splanchnic nerve would bring about a vaso dilation of the renal vessels and cause a reduction of blood pressure in hypertensive disease, the Department of Surgery at the University of Michigan Hospital has performed the technique of splanchnicectomy on more than 200 patients. The operative procedure calls for a bilateral resection of the greater, lesser and least splanchnic nerve, and excision of the tenth, eleventh and usually the twelfth thoracic sympathetic ganglion. The first operations were done in two stages, first the right and then the left side, but more recently the bilateral operation has been performed at one sitting, the complete procedure requiring approximately one hour.

It is too early to predict the value of splanchnicectomy in hypertension, although some remarkable results have been observed. Many of the patients have experienced an appreciable drop in blood pressure with disappearance of headache, vertigo, nervous irritability and tinnitus. It is possible that we are dealing with an operative procedure which, when it causes a reduction in blood pressure, may arrest the progress of deafness in essential hypertension and in some cases bring about improvement through the elimination of tinnitus and throbbing noises in the head. The effects of splanchnicectomy as revealed by a study of 156 patients are gratifying enough to inspire our continued interest and enthusiasm in this work.

Symposium on Essential Hypertension

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THE NATURE AND SIGNIFICANCE OF THE RETINAL LESIONS ASSOCIATED WITH HYPERTENSIVE DISEASE

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THE FULL significance of the lesions of the retina and its vessels which occur among individuals who have elevated blood pressure will be known only when their detailed pathogenesis is definitely established, probably through experimental reproduction of these lesions. At this time, the retinal pictures typical of chronic or rapidly progressive hypertensive disease have not been reproduced in characteristic form or course in experimental animals. The lesions observed by Keyes and Goldblatt in the eyes of animals that had elevated blood pressures resulting from constriction of the main renal arteries simulated in certain respects those seen in association with hypertensive disease in man. Yet, certain differences are present which are thus far difficult to explain. In spite of the inadequacy of present experimental evidence, however, advances are being made in the understanding of the mechanisms involved in the production of the retinal lesions and of their relationship to the systemic, pathologic, and pathologic-physiologic processes of the hypertensive individual.

The conception that a toxic substance circulating in the blood is responsible for the lesions in the retina commonly designated as "retinitis" and that the kidneys are the sources of this toxic substance has been most widely accepted. It has been impossible thus far to demonstrate the existence of such a toxic substance. Clinical investigation has demonstrated conclusively that the usual retained nitrogenous waste products associated with impaired renal

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function cannot be held responsible for the development of the "albuminuric" type of retinitis. The most recent exponents of the toxic theory and its support by animal experimentation, Koyanagi and his co-workers, especially Yonechi, have produced retinal edema by the intracarotid injection of a nephrotoxin which has its primary effect apparently on the retinal pigment epithelium. Koyanagi believes that in clinical albuminuric retinitis also, the primary lesion is in the pigment epithelium with the production of subretinal edema to which all the other lesions in the retina and its vessels are secondary.

Keyes and Goldblatt demonstrated among dogs and monkeys that vascular lesions may develop independent of retinitis, presumably primarily through the effect of a vasopressor substance formed in the ischemic kidney and later intensified, perhaps, by the mechanical effect on the walls of the vessels of the elevated blood pressure itself. Even these workers believed, however, that the presence of severe renal insufficiency is necessary for the development of extensive retinitis and papilledema. They did not describe the mechanism by which the renal insufficiency induces these severe retinal lesions.[8]

Clinically, it seems obvious that the most severe forms of retinal vascular disease and of retinitis can develop in the course of hypertensive disease in the absence of any marked or even definite evidence of renal impairment. When severe renal insufficiency occurs in association with retinitis in patients who have primary hypertension, it seems probable, as emphasized recently by Page, that the damage to the kidneys is not the essential feature of the disease but that it is only a part of widespread injury to the vascular system. It has been shown by Wilson and Byrom that, in rats, constriction of the main renal artery to one kidney may result in a malignant form of hypertension with the development of necrotizing arteriolar lesions in the arterioles of the other kidney as well as in other organs of the body. They expressed the opinion that the changes in the arterioles were due to the vascular strain imposed by a rapidly developing hypertension and that renal failure played no part apparently in their origin. That the presence of a primary involvement of the kidneys is not essential to the production of an "albuminuric" type of retinitis is demonstrated especially by the fact, noted by Volhard[35] and by Keith and me, that retinitis indistinguishable from the usual type, both in its vascular and retinal lesions, may develop in the course of hypertension asso-

ciated with basophilic adenoma of the pituitary body (Cushing's disease), or with adenomas of the adrenal cortex or tumors of the medulla of the adrenal glands.

In cases of "albuminuric retinitis," fairly widespread sclerosis of the arterioles has been demonstrated to be present in the retina at necropsy by Fontaine, Verwey, Kyrieleis, Friedenwald, and Gasteiger. It seems unlikely, however, both from the clinical and histologic standpoints, that organic damage to the retinal vessels can be held responsible for the development of the forms of retinitis characteristic of hypertensive disease. It may be possible, as suggested by Friedenwald, that the "toxic substance" demanded by many authors for the production of the retinal lesions may have its origin not in the kidneys but locally in the retina as the result of nutritional disturbances of tissue consequent to impaired circulation. In any event, it seems most logical at present to assume that retinitis develops in some manner as a form of decompensation of the retinal circulation. Whether this decompensation is the expression in the retina of the ischemia of Schieck[31,32] and of Volhard,[36,38] the passive venous hyperemia of Mylius, the anisoperfusion of Fritz, or of some other mechanism as yet unexplained, cannot be decided on the basis of the available data. In all events, however, it seems most probable that the decompensation results from functional disturbances of circulation and not from organic lesions in the walls of the vessels.

In the clinical study of the retina of individuals who have elevated blood pressure, the changes in the arterial vessels which precede the development of lesions in the retina proper are of great importance. The presence and nature of these changes are not explained as a rule by those who favor the toxic origin of the retinitis. If we assume that the retinitis in itself is merely an expression of decompensation of the retinal circulation, it becomes evident that, in the entire group of cases, the significance of ophthalmoscopic studies of the retina lies predominantly in the interpretation of the processes going on in the retinal vessels as a part of the general vascular system. From the purely clinical standpoint this seems to be the only logical way to explain the participation of the retina in the various types of disease in which elevation of blood pressure is encountered. It becomes obvious, then, that detailed study of the changes observed ophthalmoscopically in the retinal vessels is of primary importance. Histologic studies of the retinal vessels reveal only the end stages of a process which, through much of its course, has no histologic counterpart.

Bailliart and his followers believed that estimation of the blood pressure in the central retinal artery is of more importance in the study of hypertensive disease than is the detailed ophthalmoscopic estimation of the condition of the retinal vessels. Certain facts with reference to the retinal arterial diastolic pressure are undoubtedly of considerable interest, for example, Suganuma's[33,34] observation that the diastolic pressure in the central retinal artery rises disproportionately to the systemic pressure in certain cases of toxemia of pregnancy. Recently, Koch has shown that a similar disproportionate rise in retinal arterial diastolic pressure occurs among hypertensive individuals who have retinitis. Bailliart has assumed that on the average, the retinal arterial diastolic pressure is 45 per cent of the brachial arterial diastolic pressure. In Koch's series, the retinal arterial diastolic pressure averaged 34 per cent of the brachial diastolic pressure. Among hypertensive patients having retinitis without edema of the disks, the percentage of retinal to brachial pressure rose to 54 and in patients with retinitis with edema of the disk, rose to 62. The significance of these observations is not entirely clear. The disproportionately high pressure in the central retinal artery may be connected in some way, either as cause or effect, with the local decompensation of retinal circulation previously postulated. Or it may be the result of the rise in the pressure of the cerebrospinal fluid noted by Pickering in cases of this type. Such studies are of importance and should be continued. But it is well known that single readings of blood pressure are of no great significance in the estimation of the type or severity of hypertensive disease. The variations of blood pressure in hourly readings over a twenty-four hour period are much more significant and further studies of retinal arterial pressures should be carried out, using twenty-four hour readings. It would be important to know also whether the disproportionate pressure in the retinal arteries is present at the lowest as well as at the highest level of the systemic blood pressure in a given individual.

Perhaps of first importance in the interpretative study of the retinal arterial vessels is the decision as to whether they should be regarded as arteries or arterioles. No definite anatomic distinction exists between arteries and arterioles. Arterial vessels of any size are differentiated from capillaries and veins by the presence of an internal elastic lamina and of muscle in the medial coat. The distinction between arteries and arterioles is largely an arbitrary one based on the diameter of the vessel. In their studies on the arteriolar lesions in hypertensive disease, Kernohan, Keith and their

co-workers considered as arterioles vessels varying in diameter from 25 to 125 microns.[14] Other authors place the upper limit of arterioles, in respect to diameter, at 150 microns. The estimated average size of the retinal arterial vessels at points just beyond the margins of the optic disk by various methods of measurement during life has been stated to be 90 microns (Neame), 90 to 112 microns (Badtke), 110 to 120 microns (Lo Cascio), 63 to 116 microns (Koch), and 88 to 134 microns (Lobeck).

Considering separately each of the main branches of the central retinal artery, Koch has found that in varying age groups the mean caliber of the inferior nasal branch varies from 63 to 70 microns, of the superior nasal branch, from 70 to 79 microns, of the superior temporal branch, from 91 to 110 microns, and of the inferior temporal branch from 101 to 116 microns. It would appear, then, that all the branches of the central retinal artery visible ophthalmoscopically in the retina fall within the size limit of arterioles and should be so designated. Lesions observed in the retinal arterial vessels should be considered as indicative of systemic arteriolosclerosis, rather than of arteriosclerosis. In a study of vascular lesions observed in the kidney, Bell has been inclined to confine the designation of arterioles to vessels of 25 to 40 microns in diameter. These are really the precapillary arterioles in which the muscle and internal elastic lamina have been reduced to a minimum. Moritz and Oldt were inclined to deny the significance of Bell's distinction in the histologic study of arteriolosclerosis in the kidneys and other organs and tissues. In general, the reaction of the arterioles to elevation of blood pressure is one of contraction of the lumen and ultimate hypertrophy of the media. Because of the minimal thickness of the media in the precapillary arterioles, Kernohan[16] thought that the reaction of the precapillary arterioles to elevation of blood pressure was one of subintimal fibrosis rather than of muscular hypertrophy of the media, and that these minute vessels might even tend to dilate rather than constrict as the result of the lowering of blood pressure distal to the point of maximal constriction in the main arterioles. Ophthalmoscopically, dilatation of small arterial vessels ordinarily not seen can be observed in some cases distal to points of extreme constriction in the arterioles. The necrotizing lesions of the arterioles which were considered by some authors, notably Fahr, as pathognomonic of malignant hypertension or malignant nephrosclerosis, were regarded by Kernohan as only the natural end stage of hypertrophy of the wall and

reduction of circulation in the hypertrophied wall. Such lesions are not confined to the malignant phase of primary hypertension, but may be seen even in cases of chronic glomerulonephritis.

In general, the changes observed ophthalmoscopically to occur in the retinal arterioles in association with hypertensive disease may be grouped under five main subdivisions: (1) generalized smooth narrowing of lumen, (2) changes in the color, reflex stripe, and visibility of the walls, (3) arteriovenous compression, (4) irregular localized constrictions of the lumen, and (5) periarterial sheathing. In many cases, various combinations of these changes are seen. Each of them can occur alone, however, and the significance of each should be considered separately.

If the color changes consequent upon changes in the blood itself are excluded, such changes as are observed in the presence of anemia, leukemia, lipemia and cyanosis, it seems probable that changes in the color of the wall of the vessel (increased brightness, "copper wire" color, and "silver wire" color) and increased visibility of the wall itself are due to an actual structural lesion in the wall, which is most often hypertrophy of the media. Appearances of distortion and compression of the veins at the points of arterial crossing have been demonstrated to be caused by thickening of the walls of the arterioles and at times by extension of the sclerotic lesions into the walls of the veins themselves. It is obvious that these structural changes in the walls of the vessels are relatively late manifestations of hypertensive disease and that they indicate duration rather than progressive severity of the disease. Hence, they will be designated in this discussion as constituting "chronic arteriolosclerosis."

Irregularly placed, localized narrowings of the lumen of the arterioles have been demonstrated definitely in many instances to be of transitory spastic, rather than permanent organic or structural, nature. Proofs of this have been furnished by Mylius, Masters, Hallum, myself and others. The ultimate proof of the spastic nature of any localized narrowing rests, of course, on its complete disappearance. In rapidly advancing or active hypertensive disease, such as that seen in association with acute toxemias of pregnancy, shifting in the situation and degree of these localized regions of narrowing can be observed from day to day. As the hypertension becomes more fixed, the spastic narrowings may become more fixed in situation also. In the more chronic forms of primary or secondary hypertensive disease, the localized narrow-

ings seem to be more persistent under the usual conditions of observation. It seems probable that the persistence of localized constriction of an arteriole at any point will result ultimately in structural changes in the wall of the vessel with permanent narrowing and sclerosis of the arteriole at that point. Whether the sclerosis thus induced differs in type from the medial hypertrophy resulting from long-standing elevation of blood pressure has not been definitely proved. It is possible that intimal proliferation is the characteristic change produced by prolonged localized constriction of the arterioles. Just how long localized constriction can persist in a region without production of structural changes in the wall of the vessel is not known. However, I have seen such narrowings disappear completely after having watched them persist for at least several weeks.

In the present state of our knowledge, I do not believe that it is possible always to distinguish actively spastic from permanent organic localized narrowings of the arterioles at a single ophthalmoscopic examination. In a constriction which is actively spastic, the vessel wall is usually less visible at the point of excess narrowing than it is in the rest of its course. This decreased visibility may be caused at times by the presence of slight edema in the surrounding portion of the retina. A permanent, organic localized constriction in an arteriole is likely to be more clear-cut than is an actively spastic constriction. When there is a definite increase in the brightness of the arteriole and increased visibility of the wall at the point of constriction, it can be assumed, I think, that the constriction is permanent and is of organic nature. This type of vascular change will be designated as "postspastic sclerosis."

It is true, probably, that in certain individual instances, localized points of constriction may be seen in the retinal arterioles as evidence of localized endarteritis or atherosclerosis. Such regions are seen as a rule among elderly individuals, usually in the larger arterioles near the margins of the disks and usually in the absence of lesions of hypertensive type in the rest of the retinal vessels. Occasionally, an actual sclerotic plaque is visible at the point of constriction; but usually the diagnosis of such constrictions as "atherosclerotic" is established by inference only, and is not proved definitely. In the main, fewer errors will be made by considering all such localized narrowings as of spastic or postspastic nature unless the presence of an atherosclerotic plaque can be demonstrated definitely.

Localized regions of periarterial sheathing seen in the retina of patients having hypertensive disease indicate, probably in all cases, the previous presence of edema in the retina around the wall of the vessel. I do not believe that periarterial sheathing develops as a part of the chronic arteriolosclerosis associated with long-standing, mild or benign hypertensive disease. It is seldom if ever seen in the absence of localized spastic or postspastic constrictions in the arterioles. In the main, the presence of periarterial sheathing can be accepted as evidence of a previous episode of retinitis.

The nature of the generalized smooth narrowing of the arterioles seen among many individuals who have hypertension is perhaps the most difficult to interpret of any of the hypertensive changes in the retina. In certain instances of rapidly rising blood pressure, as is the case in hypertensive toxemia of pregnancy, in acute vasospastic disease with hypertension, and in some cases of glomerulonephritis, the narrowing of the arterioles may be actively vasospastic in character and is often the precursor of acute retinitis. In other cases of milder and more chronic hypertension, the narrowing may be a manifestation of sclerosis in the arterioles themselves or of sclerosis or narrowing of the lumen of the central artery in the optic nerve.

In most cases, however, the size of the arterioles in the retina is most probably an expression of vasomotor tone, and reduction in their size below that of the average normal indicates an increase in their tonicity. That this is true is suggested by the fact that the diameter of the arterioles corresponds in a general way to the height of the diastolic blood pressure readings. Thus, Koch found that in a group of individuals in which the diastolic pressure ranged from 80 to 120 mm. of mercury, the mean caliber of the retinal arterioles varied from 74 to 83 microns, as compared to the normal caliber of 88 to 94 microns. In a group in which diastolic pressures varied from 95 to 140 mm. of mercury, the mean caliber of the retinal arterioles was 68 microns, in another, with diastolic pressure of 115 to 150 mm. of mercury, the mean caliber was 60 microns, and in another in which diastolic pressure ranged from 140 to 155 mm. of mercury, the mean caliber was 46 microns. Obviously, there is a certain amount of overlapping of these figures, and members of the groups were selected on the basis of the general severity of the hypertensive disease present, rather than on the elevation of the blood pressure readings alone. Yet the figures do indicate the general tendency toward narrowing of the

retinal arterioles as the blood pressure rises. Repeated observation of individual cases demonstrates that the size of the arterioles is not constant but may fluctuate with variations in the blood pressure. Thus, the caliber of the retinal arterioles may be seen to increase after splanchnic sympathectomy for the relief of hypertension. Also, in certain cases of cardiac decompensation resulting from chronic hypertension, the arterioles of the retina are noted to be of greater diameter than they were before decompensation began. Other observers have demonstrated by measurement the decrease in the size of the arterioles in the retina associated with elevated blood pressure. Thus, in cases of the type which we would designate as "malignant hypertension," Lobeck found that the diameter of the retinal arterioles ranged from 27 to 58 microns, as compared to his normals of 88 to 134 microns, and Badtke gave as his figures 60 to 91 microns, as compared to his normals of 90 to 112 microns, with an average of 105 microns for his normals and 70 microns for his hypertensives. That the presence of a chronic medial hypertrophy type of sclerosis in the arterioles does have some effect in narrowing the lumen of the arterioles is suggested by Koch's findings in his group of hypertensive individuals. Among those persons who did not have chronic sclerosis, the mean caliber of the retinal arterioles was 81 microns; among those who had sclerosis graded 1, it was 71 microns; among those who had sclerosis graded 2, it was 63 microns, and among those who had sclerosis graded 3, it was 56 microns.

According to Volhard's[37] conception, two distinct types of primary hypertensive disease are readily distinguishable: (1) "red" hypertension, in which there is no tendency toward arteriolar constriction but rather toward arterial dilatation, and (2) "pale" hypertension, the characteristic feature of which is spastic constriction of the arterioles. According to Badtke, it is possible to distinguish these two types of hypertension by measurements of the retinal arterioles. In "red" hypertension, the mean caliber of the retinal arterioles ranges from 100 to 145 microns, according to his figures, compared to the normal of 90 to 112 microns. However, Lobeck found that in "red" hypertension the mean caliber of the retinal arterioles ranged from 74 to 127 microns, compared to the normal of 88 to 134 microns. My own general impression agrees with Lobeck's figures rather than with those of Badtke, in that I think that the arterioles of the retina are of normal or greater than normal caliber in very few individuals who have hyperten-

sion in the absence of signs of cardiac decompensation. It is true that at the clinic no effort is made to differentiate "red" and "pale" hypertension, since it is believed that in practically all cases of hypertension an angiospastic tendency is evident at times (Table 1).

Of considerable interest is a group of cases in which the patients have been admitted to the hospital with symptoms of cardiac failure with normal blood pressure readings, often without a history of previous hypertension, but in which observations at necropsy show that the cardiac failure had been on the basis of hypertensive disease. In the majority of such cases, ophthalmoscopically observed changes in the retinal arterioles are minimal and consist usually of mild evidences of chronic hypertensive sclerosis without narrowing of the caliber of the arterioles. It seems to be questionable whether these cases belong to the group described by Volhard as "red" hypertension or whether a narrowing of the retinal arterioles which had been present previously is no longer visible because of general loss of vascular tone associated with the cardiac failure.

It is generally accepted at present that reactions visible in the retinal vessels furnish a fair guide to the nature if not to the degree of the reactions taking place in the arterioles throughout the organs and tissues of the body. A detailed consideration of the changes visible in the retinal arterioles, according to the plan previously described, will enable physicians to determine whether simple increase in tone of the arterioles is being dealt with or whether this increase in tone is combined with a chronic medial hypertrophy or with a tendency to localized angiospasm or with both. It will thus be possible to judge with considerable accuracy from the appearance of the retinal arterioles alone as to the duration, type, severity, and progressive character of the hypertensive disease in individual instances.

It seems obvious from histologic as well as from ophthalmoscopic observations that the usual types of hypertensive retinitis, with edema of the retina, cotton-wool patches, and hemorrhages, are not the results of sclerosis of the retinal arterioles. The hyaline lipoid necrosis of the terminal arterioles described in these types of retinitis by Verwey, Kyrieleis, Friedenwald, and Gasteiger may be the result of the lowered pressure in these vessels resulting from extreme constriction in the more proximal and larger arterioles, or it may result from toxins generated in the retinal

tissues, as suggested by Friedenwald. In any event, it would seem to be a result or a concomitant feature of the retinitis rather than its cause. It is questionable whether the "retinitis of arteriosclerosis" as designated by Foster Moore actually exists. Actual closure of the lumen of arterioles gives rise in its acute phase to the picture of an ischemic infarct and later to atrophy of the affected part of the retina, which often can be recognized only by loss of vision in the corresponding sector of the field of vision. The picture of localized groups of punctate exudates and hemorrhages, described under the name of "retinitis of arteriosclerosis," seems in most instances to represent the end phase of venous obstruction or thrombosis. Thrombosis of a branch of the central vein or of the central vein itself is a rather frequent complication of hypertensive arteriolosclerosis in the retina. Such thrombosis is a feature of the milder, more chronic cases of hypertension rather than of the more severe and rapidly progressive types. In many instances, the sclerosis visible in the arterioles is so minimal that it may be wondered whether some focal or other type of infectious process has not been a factor in the production of the thrombosis through the introduction of complicating phlebitis. This supposition is supported in some instances by the favorable course of the thrombosis following the removal of some obvious focus of infection. In the end stage of thrombosis of a branch vein, particularly of a small one, after a certain amount of collateral circulation has been formed, punctate hemorrhages and punctate exudates residual to the localized region of edema in the retina may produce a picture which may be readily diagnosed as "retinitis of arteriosclerosis" if careful search is not made for the obstruction of the small vein which was the original cause of the retinal lesion.

A picture resembling that described as "retinitis of arteriosclerosis" may be seen also in the terminal phases of angiospastic retinitis, particularly of one in which the edema has been relatively scanty and perhaps confined to localized portions of the retina. After the originally present cotton-wool patches have been absorbed and the flame-shaped and larger hemorrhages characteristic of the early stages of the retinitis have given place to small, punctate, deeper hemorrhages, scattered individual or groups of punctate exudates may remain for a long time before complete disappearance of the retinitis takes place. In such cases which have apparently lost, for the time being or permanently, their angiospastic tendency, the association of such lesions in the retina

with a considerable grade of sclerosis in the arterioles, may lead to the diagnosis of "retinitis of arteriosclerosis." In essence, however, these retinal lesions are not the result of arteriosclerosis.

Fundamentally, the lesions in the retina which are designated as constituting "retinitis" or "retinopathy" are identical in nature, although they may differ in degree and extent, in all cases of hypertension whether of primary essential or secondary type. Their common and characteristic feature is edema in the retina and subretinal space, as has been pointed out by Koyanagi. Fundamentally also, this type of retinitis is acute in onset. If the truth of these two fundamental facts is recognized, it becomes relatively easy to understand the diagnostic and prognostic indications furnished by the occurrence of this acute retinal episode, possibly circulatory decompensation, in association with varying types of change in the retinal arterioles. Personally, I am inclined for the present to accept Volhard's[38] view that retinitis of this type is associated always with a generalized angiospastic episode and a relatively excessive elevation in blood pressure. I prefer, therefore, to use the term "acute angiospastic" to designate the types of retinitis which formerly were called "albuminuric." Acute angiospastic retinitis can occur in the presence of uniform generalized constriction of the arterioles only, in the presence of generalized and localized excess narrowing of the arterioles, or in the presence of generalized and localized narrowing with additional evidence of chronic sclerosis or medial hypertrophy of the walls of the arterioles.

Acute angiospastic retinitis without chronic sclerosis of the arterioles may be seen in hypertensive toxemia of pregnancy, in acute vasospastic disease with hypertension,[41] in acute glomerulonephritis and in chronic glomerulonephritis. In all of these conditions the elevation of blood pressure is of relatively recent origin in relationship to the development of retinitis. Theoretically, the prognosis for the life of the individual should be good in this type of retinitis, since it represents an acute, rather than a chronic, and a functional, rather than organic, involvement of the arterioles. Practically, this assumption is found to be true except in the presence of chronic glomerulonephritis. In hypertensive toxemia of pregnancy, recovery begins as soon as the pregnancy is terminated. In acute vasospastic hypertension, also, there is a definite tendency toward recovery, especially if the source of the angiospasm can be discovered and eliminated. The blood pressure tends to return to normal or to definitely lower grades. In cases in which

the tendency to angiospasm cannot be controlled, recrudescence or continuance of the angiospastic lesions in the retina leads to the development of prognostically more serious types of retinitis, with the gradual addition of chronic arteriolosclerosis.

Acute angiospastic retinitis without chronic sclerosis of the arterioles may be seen occasionally among patients who have acute glomerulonephritis. Here, also, the prognosis seems to be good. Usually, the retinitis is mild and subsides promptly. In the main, its presence does not seem to alter the prognosis of the nephritis. No definite statistics are available, however, to show whether the occurrence of such a type of retinitis has any prognostic value in the tendency of the nephritis to become chronic rather than to subside completely.

The prognosis of that type of acute angiospastic retinitis seen in association with chronic glomerulonephritis is definitely more serious. Obviously, this seriousness is due to the antecedent presence of marked damage to the glomeruli and other structures in the kidneys. The development of this type of retinitis in such cases is always associated with an elevation of blood pressure in cases in which the blood pressure had been normal previously, and with a relative excess elevation in cases in which the blood pressure had been elevated previously. The functional constriction of the arterioles which is visible in the retina must also involve the arterioles of the kidneys. As a result, there is a rapid decrease in the excretory function of the already damaged kidneys, a decrease which ushers in the terminal phase of the disease. Acute angiospastic retinitis without chronic sclerosis of the arterioles must be regarded as the typical retinitis of chronic glomerulonephritis. It was noted by Graham in thirty-one of fifty-five cases demonstrated at necropsy to be definite chronic glomerulonephritis. Since retinitis was present before death in only forty-three of these cases, the retinitis was of the acute angiospastic type without chronic sclerosis in 77 per cent of all the cases of retinitis. The mean length of life for the patients in this group after the development of acute angiospastic retinitis was 4.1 months.

When acute angiospastic retinitis occurs in the presence of chronic sclerosis of the arterioles, the prognosis for the life of the patient is always more serious. Obviously, further reduction of circulation through generalized arteriolar constriction to organs and tissues already suffering from improper nutrition through the presence of sclerosed arterioles results in more rapidly progressive

damage to the walls of the vessels themselves, as well as to the organs and tissues supplied by them. This type of retinitis can be seen in two forms, one as a relatively localized or scanty involvement of the retina without edema of the disks, and the other as a diffuse involvement of the retina with edema of the disks. Both these types of retinitis are definitely an indication of a "pale" or angiospastic type of hypertension. But the prognosis for the survival of the individual is definitely worse in the presence of retinitis with edema of the disks. In the type of retinitis without edema of the disks, there is a definite tendency for at least temporary subsidence of the retinitis and of the angiospastic episode. No such tendency is apparent in the case of retinitis with edema of the disks, except in very rare instances. The course of the hypertensive disease among individuals who exhibit retinitis with edema of the disks and chronic sclerosis of the arterioles is usually steadily progressive toward a fatal termination. Hence, this type of retinitis is often designated as the "retinitis of malignant hypertension." Hypertensive disease among individuals having acute angiospastic retinitis and chronic sclerosis of the arterioles, but without edema of the disks, is also of serious and progressive character. However, it advances usually in a series of recurrent angiospastic episodes and not with the steadily progressive course of the typical "malignant" cases.

Should an individual have acute angiospastic retinitis with chronic sclerosis of the retinal arterioles, it means always that the individual has diffuse arteriolosclerosis, no matter whether his primary disease is "essential hypertension" or "chronic nephritis." As has been shown, the retinitis itself cannot be considered to be caused vaguely by the "nephritis" or by some unknown "toxin" of renal origin, or by retained products of renal insufficiency. Logically, the retinitis can be regarded only as a manifestation of decompensation of the retinal circulation. Chronic sclerosis of the arterioles in the retina can be demonstrated definitely to be a part of diffuse sclerosis of the arterioles in various organs and tissues throughout the body. Thus, Graham found diffuse angiospastic retinitis with chronic sclerosis of the arterioles in eight of his fifty-five cases of chronic glomerulonephritis. In all of these eight cases there was a marked sclerosis of the arterioles in the kidney which could not be differentiated histologically from that seen in cases of malignant hypertension or malignant nephrosclerosis. On the other hand, renal arteriolosclerosis was present in only three of

the thirty-one cases of acute angiospastic retinitis without chronic sclerosis of the retinal arterioles; in these three, it was mild in degree. It seems logical to assume, then, that lesions in the retinal arterioles are not a part of the "nephritis" proper but are an indication of primary or complicating arteriolar disease, as the case may be.

At the 1932 meeting of the American Academy of Ophthalmology and Otolaryngology, Keith[12] called attention to his division of patients having hypertensive disease into four numbered groups for purposes of differential diagnosis and prognosis. The definite value of this division has been demonstrated by further study. Among a large number of patients who have essential hypertension, the elevation of blood pressure may be present for a considerable time without producing definite symptoms and without impairing cardiac or renal function. Such individuals are placed in group 1 of diffuse arteriolar disease with hypertension. The retinal arterioles of these patients rarely exhibit more than a mild grade of narrowing and chronic sclerosis. Patients who are diagnosed as having diffuse arteriolar disease with hypertension group 2 have a more sustained and usually higher elevation of blood pressure than those in group 1 and are more likely to have symptoms such as increased nervousness and headache. The retinal arterioles exhibit a correspondingly higher grade of organic vascular disease. Group 3 of diffuse arteriolar disease with hypertension includes patients who show subjective and objective symptoms similar to, but often more severe than, those observed among individuals belonging to group 2 and, in addition, definite evidences, especially in the retina, of the occurrence of angiospastic episodes. The only definite criterion established as yet for the inclusion of an individual at the first examination in group 4 of diffuse arteriolar disease with hypertension is the presence of diffuse angiospastic retinitis with chronic sclerosis of the arterioles and with measurable edema of the optic disks. Although subjective symptoms are likely to be severe among these patients, it is surprising how nearly symptom-free some of them are. As a rule, the diastolic arterial pressure reaches higher values than it does in the other groups, and is sustained at a high value over the entire twenty-four hour period. Cardiac and renal functions are often entirely adequate at the time the diagnosis is established, but usually become definitely impaired in the later course of the disease.

The separation of patients who have hypertensive disease into these groups is of particular value from the standpoint of prognosis of life expectancy. Keith[40,41] made a follow-up study of 219 patients who had hypertensive disease for a period of five to nine years after initial examination. The time limit at which 50 per cent of the patients had died was found to be 100 months in group 1, sixty-three months in group 2, 15.9 months in group 3, and 5.4 months in group 4. Again, of ten patients assigned to group 1, six were alive seven years later (60 per cent); of twenty-six patients in group 2, nine were alive six years later (34 per cent); of thirty-seven in group 3, three were alive six and a half years later (8 per cent); and of 146 assigned to group 4, only one was living five years later (0.6 per cent). The mean length of life was found to be 27.6 months for patients belonging to group 3, and 10.5 months for patients assigned to group 4. Seventy-nine per cent of the patients diagnosed as having diffuse arteriolar disease with hypertension group 4 died within a year of the time that the diagnosis was established.

The importance of careful study of the retina and its arterioles in the classification of these patients is obvious. For essentially, the characteristic feature of members of groups 1 and 2 is slowly progressive organic disease of the arterioles, whereas in groups 3 and 4 evidence of active constriction or spasm of the arterioles is outstanding. Patients who have purely chronic lesions in the arterioles of the retina will be placed usually in groups 1 and 2, even when sclerosis is complicated by the presence of venous thrombosis or of the "retinitis of arteriosclerosis" on the basis of previous venous thrombosis. It is true that certain patients having visible localized narrowings of the retinal arterioles are included at present in group 2 if no definite proof of activity of the causative spasm can be elicited and the lesions are considered to be postspastic sclerotic in character. In such cases, however, careful watch should be kept for the possible recurrence of active spasm. For patients who exhibit evidence of active angiospasm in the form of retinitis, the prognosis is always serious. It is not quite so serious, however, for individuals who have relatively patchy angiospasm as evidenced by the presence of scattered cotton-wool patches and hemorrhages as it is for patients who have diffuse retinitis with edema of the optic disks. In the case of the patient who has retinitis of diffuse arteriolar disease with hypertension group 3, remissions may and frequently do occur. But for the

other patient, who has retinitis of diffuse arteriolar disease with hypertension group 4, the possibility of remission is extremely remote.

It must be remembered that these groups do not represent fixed entities or basically distinct types of hypertensive disease. A patient whose disease at the time of the initial examination would seem to belong to group 1 or 2, may at a later date show definite evidences of an added angiospastic factor and may have to be placed in group 3 or 4. On the other hand, in the case of certain individuals whose condition would be classified initially as belonging to group 3 and very occasionally as belonging to group 4, the angiospastic features may disappear and the hypertensive disease may revert to group 2 and even to group 1. The sliding nature of this grouping is particularly valuable and necessary at present and will probably continue to be so until more is known about the basic etiologic factors of the hypertensive disease and its structural and functional arteriolar lesions.

SUMMARY AND CONCLUSIONS

The changes visible ophthalmoscopically in patients who have elevation of blood pressure are primarily demonstrations of disturbed physiology of the arterioles, whether the hypertension is of the so-called primary type or is secondary to inflammatory renal or endocrine disease. The lesions in the arterioles are always primarily, and possibly in large part at all times, of functional nature. Histologically demonstrable lesions in the arterioles are always a late feature of the disease and may be absent at necropsy, even in the presence of retinitis. At present no definite etiologic relationship can be established between retinitis and any histologically demonstrable lesion in the parenchyma of the kidney or between retinitis and any demonstrable circulating product of disturbed renal metabolism. Sclerosis of the arterioles of the retina can be demonstrated definitely to be a part of diffuse sclerosis of the arterioles throughout the body. On the basis of present knowledge, the retinitis which occurs in association with elevation of blood pressure can be explained logically only as being a manifestation of some form of decompensation of the retinal circulation.* If the kidneys have any direct influence in the development of retinitis of this type, it must be through the elaboration of a thus-far hypothetic effective substance.

* Although this conception cannot be assumed to be definitely proved at present, yet it provides at least an adequate working hypothesis.

TABLE 1 — *Measurements of Retinal Arterioles in Microns*

	LOBECK	LO CASCIO	NEAME	BADTKE	KOCH
Normal	88-134	110-120	90	90-112	63-116
Red hypertension	74-127			100-145	57-102
Pale hypertension	53-81			60-91	35-70
Transition form				60-85	50-84
Malignant nephrosclerosis with albuminuric retinitis	26-33				35-55
Chronic nephritis	53-62				51-96
Chronic nephritis with normal diastolic blood pressure				96-125	72-96
Chronic nephritis with elevated blood pressure					51-83

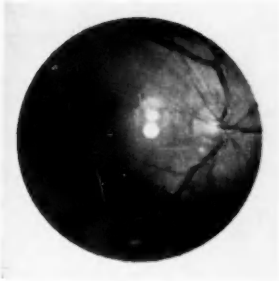

FIG. 1 — (Wagener) — Chronic sclerosis of retinal arterioles.

FIG. 2 — (Wagener) — Postspastic sclerosis of retinal arterioles.

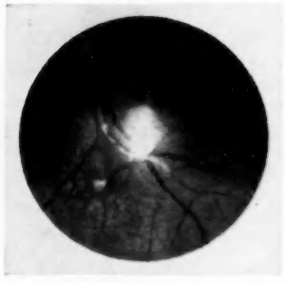

FIG. 3 — (Wagener) — Periarteriolar sheathing in recurrent angiospastic retinitis; diffuse arteriolar disease with hypertension, group 3.

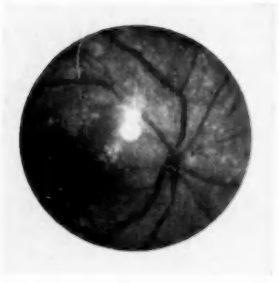

FIG. 4 — (Wagener) — Acute angiospastic retinitis without chronic sclerosis of retinal arterioles (residual phase); hypertensive toxemia of pregnancy.

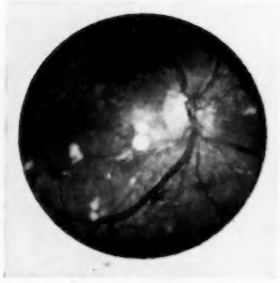

FIG. 5 — (Wagener) — Acute angiospastic retinitis with chronic sclerosis of retinal arterioles without edema of disks; diffuse arteriolar disease with hypertension, group 3.

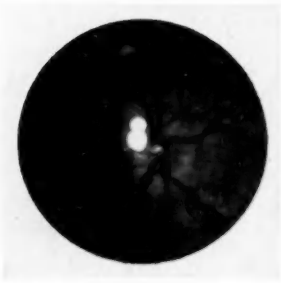

FIG. 6 — (Wagener) — Acute angiospastic retinitis with chronic sclerosis of retinal arterioles and edema of disks; diffuse arteriolar disease with hypertension, group 4.

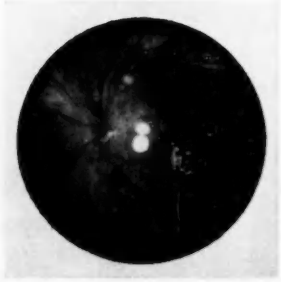

FIG. 7 — (Wagener) — Acute angiospastic retinitis with chronic sclerosis of retinal arterioles and edema of disks; chronic glomerulonephritis with complicating diffuse arteriolar disease.

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Symposium on Essential Hypertension

* * *

ESSENTIAL HYPERTENSION FROM THE STANDPOINT OF THE INTERNIST

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WE MAY define the condition most widely known as primary or essential hypertension, as one in which there is a persistent elevation in both the systolic and diastolic blood pressures, unrelated to primary renal disease, endocrine or vasomotor disorders. It is, by all odds, the most common type of hypertension encountered in medical practice and is of particular interest to the internist, because it is a symptom so often found in a large number of indiduals over forty years of age, who ultimately succumb to heart failure, a cerebral accident or renal insufficiency. Since more individuals are reaching the later decades of life than ever before, essential hypertension, like cancer, is increasing in frequency, and is now one of the most common problems with which the internist has to deal.

THE PATHOGENESIS OF ESSENTIAL HYPERTENSION

Until quite recently, the etiology of essential hypertension has been wrapped in obscurity, but thanks to the classic experiments of Goldblatt and his associates, facts have replaced fancies and the shadows hitherto surrounding the pathogenesis of essential hypertension are gone. I have followed Goldblatt's work since its inception some ten years ago in the Pathological Institute at Western Reserve, and believe that it affords the first clear demonstration of the renal origin of arterial hypertension.

In interpreting his observations, I am not unmindful of the limitations imposed in applying the results of animal experimentation to the problem of human hypertension, but in the face of such

clear-cut facts the risk of speculation is greatly reduced. Goldblatt's experiments have been repeated and verified by many workers both here and abroad, and since the results appear to be a landmark in our knowledge of the subject, it seems advisable to present at this time, a brief resume of our changing conceptions during the past century regarding the relationship of hypertension, kidney disease and arteriosclerosis.

With no means of determining clinical blood pressure, Richard Bright in 1836 postulated an abnormal peripheral resistance in the arterioles and capillaries to account for the thickened arteries and hypertrophied hearts, which he found so frequently at post mortem in patients exhibiting scarred kidneys. Believing that the changes in the heart and arteries were due to kidney disease, it may therefore be said that Bright was the first to consider the renal origin of hypertension. George Johnson in 1868 confirmed Bright's observations and agreed that cardiac hypertrophy, as well as diffuse vascular sclerosis, was secondary to renal disease. A dissenting opinion appeared in 1872, when Gull and Sutton, impressed by the diffuse vascular lesions which they called "arterio capillary fibrosis" said: "These changes are, or may be independent of renal disease, and that the renal change in chronic Bright's disease with contracted kidneys, when present, is but a part of a general morbid condition." In thus concluding that diffuse vascular disease is a primary pathologic entity responsible for increased arterial pressure, Gull and Sutton founded a school which stressed the nonrenal origin of hypertension, a conception which as we shall see, has influenced accepted views even to the present day.

With the introduction of the sphygmomanometer, it was natural that attempts were made to correlate hypertension with arteriosclerosis, but exceptions soon appeared, and in 1893 von Basch after observing many cases of hypertension with no demonstrable changes in the arteries, concluded that the elevation in blood pressure was but a precursor of arteriosclerosis. Impressed by the large number of hypertensive patients who did not have widespread vascular disease, Huchard in France and Albutt in England concluded that hypertension was due to a generalized vasoconstriction. There remained the question of what caused the vasoconstriction, but the kidney was not seriously considered as playing a major role in the production of this type of hypertension.

Up to the turn of the century there were three schools of thought concerning the interrelation of hypertension, arterio-

sclerosis and renal disease: (1) the followers of Bright, who believed that hypertension was due primarily to renal disease, (2) the followers of Gull and Sutton, who ascribed hypertension to widespread vascular disease, and regarded the renal lesions as purely secondary, and (3) those who believed with Albutt that hypertension was due to generalized vaso-constriction unrelated to renal disease. As yet no clear distinction between the various types of kidney disease, as we now know them, had been made. Kidney lesions were described as "large and smooth" and "small and granular," exceptions being regarded as intermediate stages in the process of development from the large to the small kidney. It was not until 1904, due largely to the work of Jores, that two distinct types of contracted kidneys were recognized, the inflammatory and the arteriosclerotic. Thus appeared an anatomic basis on which the clinician might distinguish nephritic hypertension — the elevation in blood pressure that is often associated with primary inflammatory kidney disease such as acute and chronic glomerulo nephritis. There remained, however, a much larger group of hypertensive patients who died of heart failure or of a cerebral accident without evidence of renal insufficiency. This fact has always been a barrier to the view that the hypertension in such cases was of renal origin.

A great advance in this field was made in 1914 by Volhard and Fahr, who showed that the kidneys of patients with essential hypertension were scarred as a result of more or less diffuse sclerosis of the smaller renal arteries, and the term nephrosclerosis was introduced to designate the condition. Volhard and Fahr recognized two general types of essential hypertension (1) the benign which pursued a chronic course without evidence of renal excretory insufficiency and (2) the malignant, which progressed rapidly to renal failure with death in uremia.

From 1914 to the present, both clinicians and pathologists have recognized that individuals with essential hypertension have pathologic changes in the smaller renal vessels in a high percentage of cases in which necropsy is done. For example, Bell and Clawson in 1928 reported observations on 420 autopsied cases of essential hypertension in which renal arterial disease within the parenchyma of the kidney was found in 97.6%, and sclerosis of the afferent glomerular arterioles in 89.4%. In spite of these results Bell and Clawson stated: "We do not have any theory of the etiology of primary hypertension to propose." As late as 1934, Fishberg in his

book on hypertension and nephritis, expressed the commonly accepted view regarding the non-renal origin of essential hypertension, when he stated: "Hypertension, like fever, is merely a symptom and not a disease; sometimes hypertension is produced by renal disease, but more often it is not."

Coming now to the present day concepts of clinical hypertension as presented in most textbooks on the subject we may summarize them as follows:

(1) Hypertension admittedly of renal origin, associated with glomerular nephritis, urinary obstruction, polycystic kidney disease, periarteritis nodosa with marked renal involvement and severe renal amyloidosis. With clear evidence of primary kidney disease in such cases, the renal origin of the hypertension is generally accepted.

(2) Essential hypertension, as we have seen, is not generally believed to be of renal origin because (a) There is no demonstrable renal excretory insufficiency in the majority of cases (b) Most patients with essential hypertension die of heart failure or a cerebral accident without ever showing renal insufficiency (c) Occasionally a case of essential hypertension is reported as exhibiting no anatomic evidence of renal arteriolar disease.

Let us now examine the foregoing arguments advanced to support the hypothesis of the non-renal origin of essential hypertension in the light of Goldblatt's experiments which show clearly the role of the kidney in arterial hypertension.

Aware of frequency of renal vascular disease in human cases of essential hypertension, and considering the possible functional effects of the process in causing progressive renal ischemia, Goldblatt in 1928, designed his animal experiments to impair gradually the blood supply to the kidneys. Unable to narrow experimentally the million or more afferent glomerular arterioles as happens in human arteriolar nephrosclerosis, he produced ischemia of the kidney by constricting the main renal artery with a special clamp so designed that any degree of narrowing up to complete occlusion of the artery could be produced. He has shown that constriction of the renal artery of one kidney in both the dog and the monkey causes an elevation in the systolic and diastolic pressures which lasts for weeks or months, whereas, narrowing of both renal arteries at once or after an interval results in persistent hypertension, which has continued in some animals for eight years. The elevated blood pressure following the constriction of one renal

artery promptly returns to normal on either removal of the ischemic kidney or release of the clamp on the artery — a crucial experiment illustrating the role of the kidney in raising blood pressure. Chronic hypertension is observed in animals that show no evidence of renal excretory insufficiency. This is the experimental counterpart of so-called benign essential hypertension in man. Also the picture of malignant hypertension with uremia, including widespread arteriolar necrosis, can be produced almost at will by further narrowing of the renal arteries. In other words, a dog with benign hypertension lasting for several years may be thrown into the malignant phase with uremic death in a few days; or the acute malignant phase can be produced in the beginning by severe constriction of both main renal arteries. In these experiments is seen the same baffling picture which has puzzled clinicians for the past hundred years, namely, the occurrence of hypertension not only with, but without renal excretory insufficiency; yet the elevation of blood pressure in both instances is clearly of renal origin. Thus the argument that essential hypertension cannot originate from the kidney because there is often no accompanying impairment of renal excretory function is no longer valid.

The failure to find renal arteriolar disease in a few persons with essential hypertension is often cited as evidence that elevated blood pressure has nothing to do with the kidney, but the possibility of severe sclerosis of the main renal arteries sufficient to cause renal ischemia has not been excluded. Up to the present such cases have been overlooked and classified as instances of essential hypertension without renal arteriolar disease.

The experimental hypertension in animals without demonstrable evidence of renal excretory insufficiency is the experimental analogue of benign hypertension commonly seen in patients. Similarly, when the renal arteries of these animals are still further constricted uremia develops with extensive retinal lesions, and post-mortem examination shows arteriolar necrosis like that observed in malignant hypertension in man. For the production of arteriolar necrotic lesions in the animal two factors are essential. (1) hypertension and (2) progressive renal insufficiency — the same combination that always occurs in the malignant phase of essential hypertension in man.

Whether the hypertension associated with primary renal disease also results from renal ischemia is a problem for the future to disclose, but it is likely that such disease of the kidney may so

alter its circulatory dynamics that the functional effects on systemic blood pressure may be the same as in primary vascular disease.

All the experiments bearing on the pathogenesis of the hypertension induced by renal ischemia indicate that it is not of reflex nervous but of humoral origin. Some chemical substance circulating in the blood and formed as a result of renal ischemia constricts the peripheral arterioles and thus elevates the blood pressure. By the same mechanism, human essential hypertension is best explained. In support of this view is the recent work of Prinzmetal and Wilson and of Pickering, who have demonstrated that essential hypertension in man is not of vasomotor origin.

It appears that renal ischemia caused either by narrowing the main renal artery or by arteriosclerotic involvement of the smaller renal vessels — the arterioles — leads to systemic hypertension. Since Goldblatt's demonstration of the role that the kidney plays in causing arterial hypertension, several cases of human hypertension have been reported in which the renal artery was narrowed or completely occluded and in which upon operative removal of the ischemic kidney, the blood pressure returned to normal.

Although Goldblatt's work throws no light on the nature of human arteriosclerosis, it goes far in clearing up the problem of hypertension, which has baffled clinicians for decades. It is now possible to understand why patients exhibiting sclerotic changes in the vascular system may or may not have arterial hypertension: if the renal vessels are sufficiently involved in the process, hypertension appears; if they are spared, the patient may show no significant elevation in blood pressure, in spite of widespread arteriosclerosis elsewhere in the body. Furthermore, it now appears that the clinical course pursued in hypertension is determined primarily by the progress of the vascular disease in the kidneys. This in the majority of cases is sufficient to cause chronic hypertension, but not rapid enough to impair renal excretory function. Thus most hypertensive patients die of heart failure or of a cerebral accident before uremia develops. However, the renal vascular changes may progress to a point when the clinical picture of so-called malignant hypertension with uremia appears. In other words, the progress and extent of vascular disease in the kidneys determine whether essential hypertension runs a benign or a malignant course.

Concluding this discussion of the etiology of essential hypertension, I will say that the evidence, both experimental and clinical, supports the hypothesis that the condition hitherto called primary

or essential hypertension is of renal origin. Arterial and arteriolar sclerosis of the renal vessels leads to renal ischemia, which by a humoral mechanism produces an increased muscular tone in the peripheral arterioles and thus raises the systemic blood pressure.

Looking back over the past century we see Bright's original postulate regarding the renal origin of hypertension, at first accepted, then questioned and finally in recent years, largely rejected. Now as a result of Goldblatt's epoch-making work, the pendulum swings back, the kidney assumes the major role in the etiology of essential hypertension and Bright stands vindicated, although it has taken more than a hundred years to do it. Thus slowly our knowledge advances. May we pause here to mention the interesting fact that the significant milestones in our understanding of blood pressure have appeared at intervals of approximately a hundred years. In 1626 Harvey discovered the circulation of the blood, in 1733 Hales first showed that blood in mammalian arteries was under considerable pressure, in 1836 Bright postulated and in 1932 Goldblatt demonstrated, the renal origin of hypertension.

Accepting the view that human essential hypertension is a symptom of renal vascular disease, the question naturally arises: what causes the sclerotic changes in the kidney vessels? Since these changes differ in no fundamental way from those seen in other arteries, our problem is, in reality, that of human arteriosclerosis. Here we stand on the threshold, three centuries after Harvey, facing in almost complete ignorance the greatest unsolved problem in medicine today. As Albutt once said, "We are groping for something in the dark."

Statistics from many sources point to the steadily mounting incidence of vascular disease, and show that it is killing more than twice as many people as any other disease. It is, at the present time, the chief barrier to longevity and the major factor determining individual expectancy. How often we see individuals in the prime of life struck down by vessel decay. It is exacting a formidable toll in our own ranks in the form of coronary thrombosis, hypertensive heart disease, cerebral hemorrhage and thrombosis. In the face of such facts we strive to explain them, only to find that our ignorance of this most common affection far overshadows our knowledge. Hypotheses there are in abundance, but these frequently express opposite views concerning the cause and nature of arteriosclerosis. In a critical review of the subject McCallum stated: "Arteriosclerosis is one of those diseases difficult to explain

because it develops so slowly through long years of life, during which a great many possible causes have had an opportunity to affect the tissues. Hence every conceivable idea has been expressed or tenaciously maintained regarding the condition, and some of them are so vague and ill supported that it is wearisome to discuss them. None, however, is clearly demonstrated to have a definite bearing on the etiology of arteriosclerosis and we are quite as ignorant of its underlying causes as were our fathers in the days of Morgagni." Obscure as the etiology of human arteriosclerosis is, clinical experience and that refined form of it we call statistics, indicate the important role that heredity plays in determining the wearing quality of our arteries.

An inherited pre-disposition to early vascular disease and a familial tendency to essential hypertension in many cases, are well established. As more people are now surviving to the later decades of life than ever before, and as more weaklings are reaching maturity to propagate their inferior stock, one sees little hope either of preventing the disease or of reducing its mounting incidence in the future.

In conclusion, may I make a few remarks on the management of the patient with essential hypertension.

We may take as a self-evident proposition, that the sound management of a sick patient depends on the physician's knowledge of the disease process responsible for the clinical picture he observes. The more accurate information we have regarding such questions as the etiology of the disease, its natural course, its amenability to any form of treatment at present available, the more rational will be the treatment the patient receives. Sound therapeutics have ever followed in the wake of advancing knowledge of disease. Our changing conceptions have often caused wide swings of the therapeutic pendulum. For example, it was not so long ago that the physician felt impelled to administer an antipyretic to lower the body temperature in many cases; now we are actually producing fever artificially, secure in the knowledge that the patient dealing with many infections fares better with a natural elevation of temperature than with one lowered by drugs. May not the same general principle apply in the treatment of essential hypertension? May not the elevation in systemic blood pressure be a natural response to guarantee a more normal circulation in such organs as the heart, brain and kidneys? Until such questions can be answered

in the negative, they merit serious consideration in any discussion of the therapeutic management of essential hypertension.

Too often our therapeutic efforts are focused entirely on ways and means of lowering the blood pressure, whereas, our zeal in this direction should at least be tempered by the evidence indicating that essential hypertension is but a symptom of renal vascular disease, which is in turn, one manifestation of human arteriosclerosis. Therefore, the therapeutics of essential hypertension is fundamentally that of arteriosclerosis.

It is well known that many hypertensive patients carry on in a normal way for years until they learn of their elevated blood pressure. Many such individuals run from one physician to another to have their blood pressure taken, and to what avail? Unnecessary restrictions of one kind or another are imposed which do no good and only remind the patient that he must be in a bad way. He may envision a stroke of apoplexy and his emotional reaction to the situation may result in a considerably higher blood pressure. It is well to emphasize, therefore, that many cases of essential hypertension not only do not need any treatment but are much better off without it. Generally the less said about the blood pressure in such people the better. Since individuals with essential hypertension deal with a chronic and at present, an incurable affliction, they are easy prey and are often victimized.

By following some course of treatment a cure may be subtly implied, if not promised. For such patients sound medical counsel is of vital importance, and in managing them, nothing quite takes the place of that homely quality we call common sense. To institute a sensible regime of living to the end that the patient exercise moderation in all things, is our first consideration. An effort should be made to dispel any phobias engendered by previous mismanagement. For example, hypertensive patients are told not to eat meat, and many develop a veritable phobia about meat in any form. Rigid protein restriction does no good and may actually cause anemia and general body weakness which disappears as the patient resumes a normal protein intake. The same may be said of all special dietaries that have been suggested for essential hypertension. Rigid salt restriction has been advocated, but no sound evidence is at hand to show that patients do any better on a salt poor diet than on one containing sufficient sodium chloride to make food palatable. To enlist the active cooperation of the patient is not always easy but none the less important. He should be told,

in words that he can understand, something of the nature of his disease, and here one should avoid reference to possible future complications such as heart failure or a cerebral accident which after all may be years in the future. The elevated blood pressure may be explained as a natural result of the changes in the arteries with advancing years, and possessing no specific remedy to put back the hands of Time, we should not promise a cure, but rather impress the patient with his own responsibility in the care of his problem.

His willingness and ability to adjust his life to whatever sensible restriction may seem indicated, may be of vital importance as the years go by. There is little to lose and much to be gained by reassuring the patient, even though we believe his future is not cheerful. Hypertensive patients, particularly women, often survive to bury their doctor.

Because of the well-known effects of suggestion in many cases, informal psycho-therapy may be tried. A daily dose of dilute hydrochloride acid caused a definite improvement in 82% of a series of carefully controlled patients with essential hypertension studied by Ayman in Boston. The symptomatic improvement following such measures, as well as the spontaneous fluctuations in blood pressure seen in many instances, forces one to be very critical in evaluating the alleged specific effects of the numerous remedies that have been suggested for the treatment of essential hypertension. Too often conclusions are reached from uncontrolled clinical observations and certain remedies are praised for a short time only to be found valueless upon more careful investigation. Such has been the story of the vast majority of remedies advocated for the treatment of essential hypertension.

Of the drugs employed at the present time, the sedatives are most often effective in controlling nervous symptoms. The relaxation induced by a good night's sleep may also cause a considerable drop in the blood pressure. In about a third of the cases treated with sulphocyanates, the blood pressure has dropped with amelioration of symptoms. However, the risk of toxic symptoms precludes the routine use of the cyanates but when controlled by regular estimation of the blood concentration of sulphocyanates, they may be given to a patient without renal impairment. The blood pressure lowered by sulphocyanates returns to its former level with the discontinuance of the drug and whether or not the patient

receives any permanent benefit from its use is a matter difficult to decide.

The latest suggestion to appear on the therapeutic horizon for the treatment of essential hypertension comes from the surgeons, who propose a variety of operative procedures designed to denervate (1) the splanchnic vessels (2) the kidneys and (3) the adrenals, with or without adrenalectomy. It seems a far cry to hope that section of nerves anywhere in the body will remove the cause or influence the course of renal vascular disease. Therefore, an operative cure for essential hypertension appears quite out of the question. However, an operation which actually increased the blood supply to the kidneys might possibly lower the systemic blood pressure and serve as a palliative measure in cases of essential hypertension.

In spite of the favorable results reported by some surgeons, we have as yet no sound evidence to show that their benefited patients live any longer or that the operation in any way retards the progress of their vascular disease. True, some patients who have submitted to one of the various operative procedures tell us they feel better, but how often we see the blood pressure rising to the preoperation level, and the patient end with heart failure or a cerebral accident, or in uremia. Until it is proved that an operation actually increases the blood supply to the kidneys, resulting in improvement of renal function in those cases in which it is impaired, the physician is on sound ground in viewing with skepticism the whole question of the operative treatment of essential hypertension.

Finally, I close this discussion by citing a proverb, "So long as more remains to be done, nothing seems done." That essential hypertension results from renal ischemia seems established, but much remains to be done before we solve that most important and most perplexing problem with which the clinician and pathologist have to deal — the problem of human arteriosclerosis.

Section on Ophthalmology

The American Academy
of Ophthalmology and Otolaryngology

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GENERAL HYPERTENSION AND PERIPHERAL OPTIC PATHWAYS

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BY INVITATION
ISTANBUL, TURKEY

I AM GREATLY honored by your kind invitation to speak before the famous American Academy of Ophthalmology and Otolaryngology. It is a favorable coincidence that the principal theme of your meeting discusses relations between general hypertension and diseases of eye and ear. As since several years I have been occupied with the study of such relations I hope to be able to add a few new points of interest.

Although science and practice have been occupied with the question of the retina and the choroid in diseases of blood pressure for many years, the part played by the optic nerve and the chiasm in these processes has been neglected.

The literature on this subject is very meager and I shall mainly make use of the results of my own researches. Not to keep your attention too long today, I want only to point out the principal results of my search, omitting nearly all references to literature and the details of my observations.

Though the amount of my examined material is large, it still is not large enough to provide certainty whether the grouping and conceptions I am now giving you are finally correct. Many more observations will have to be collected and I regard this only as a basis for further research and for discussion.

In many cases, unfortunately not in all of them, it was possible to compare the clinical with the anatomical state. The histologic examination was performed in such a manner that the optic nerve was cut into many pieces so that it was possible to examine them with different methods of imbedding and numerous methods of staining.

I begin with the processes in the *connective tissue*. Although the changes which I have observed in the connective tissue of the optic nerve have in the majority of cases no great clinical impor-

tance, I still want to mention them, since they give us certain clues, also because, perhaps, one cannot realize their importance for the understanding of the pathological occurrence in the hypertensive diseases.

I do not mean to speak much about the changes in the walls of the central artery and vein of the retina. They are doubtless frequently to be found in patients with hypertension, but have no bearing on the typical consequences of hypertensive diseases as for example retinitis albuminurica. In several cases I could observe a localized proliferation of the axial fasciculus in the neighborhood of the central artery. This apparent proliferation of connective tissue had nothing to do with an edema. I think it quite possible that it had something to do with the condition which can be brought into relation with arteriosclerosis. This point of view was maintained by Hueck, who did not limit the changes in arteriosclerosis to the walls of the vessels, but also included the surrounding connective tissue.

The microcellular infiltration in the axial fasciculus of which we have already spoken was traceable in some other observations in cases with retinitis albuminurica as well as in cases without retinal changes. It is of special interest that this infiltration was localized not around the vein but usually around the artery. The artery is, however, not always diseased. Special attention was given to the small vessels of the septa and of the Pia of the optic nerve. In systematic examination of a larger number of cases, the unusual method of imbedding paraffin was used to obtain sections as thin as possible, and in order to scrutinize the smaller vessels more accurately.

Now I want to speak about the *processes in the nerve tissue itself.*

The changes which were here to be noticed were mainly of a degenerative nature. The principal question is, however, where this degeneration develops first. It does not seem to be true that the optic nerve undergoes only an ascending degeneration which results as a consequence of the retinal process. This point of view was apparently held so far and therefore the changes of the optic nerve were regarded as uninteresting.

a) *Ascending Degeneration*

It is certain that in retinitis albuminurica an ascending degeneration of the optic nerve is possible. But on the ground of micro-

scopical research it must be admitted that only certain cases of retinitis albuminurica show such consequences in the optic nerve. Further, that these consequences are absent just when one would expect them most. The fact is really very astonishing that a papillo-retinitis albuminurica of severe character can exist for a long time and produce either no degeneration of the optic nerve or cause it late and in a mild form.

I show you first a papillo-retinitis of a 38 year old patient. The condition was present when he came to the hospital for the first time, and seven months later when he was again admitted, it was still more extensive. A severe uremia also was present — the arteries as thin as threads and the veins as if divided. The patient died from an apoplectic stroke. Microscopically there was a characteristic papillo-retinitis albuminurica with a moderate arteriosclerosis of the retinal vessels. Important for our discussion here is the state of the optic nerves.

The optic nerve trunk of the right side showed a very small limited defect, which could be traced right into the intracranial optic nerve. It is noteworthy, that this small peripheral degeneration corresponds to the nasal part of the retina, in which much less change is to be seen than in the temporal. Also in the left optic nerve part of the nerve fibers in the periphery of the retrobulbar section is lighter. Even with the Marchi staining method, which shows up the earliest stages of degeneration, no degenerations were to be seen.

Even more striking was the clinical and anatomical evidence found in a 40 year old patient, who ten years previously had some trouble with his kidneys accompanied by edema. Later on, a year ago, he had another attack and after a few months it was noticed that his visual powers had decreased considerably. Lately he has been almost blind. His blood uremia was 428 mg.%, blood pressure was elevated and very considerable quantities of albumen were present in the urine. Ophthalmoscopically an unusually severe retinitis was seen, which in parts resembled a detached retina, but had changed the whole posterior fundus into a yellowish mass with hemorrhages.

Histologically, apart from a very heavy fatty infiltration of the retina an enormous edema was found. The changes were more marked than I have ever seen. In contrast to this on one side of the optic nerve there was only a paleness of some peripheral fibre bundles, the character of which was not with certainty revealed

to be degenerative. On the other optic nerve there was a small degree of degeneration of some peripheral bundles which disappeared toward the middle of the orbital portion.

From these two cases the conclusion is justified that with a retinitis albuminurica which may even be very severe, one does not always find degenerative changes in the optic nerve. They may be completely absent or sometimes present to a mild degree. This may be so, although the condition has been present many months or even longer than a year.

The second conclusion which appears to us of interest in ascending degeneration is that in certain kinds of retinitis secondary atrophic changes occur in the optic nerve which, however, may remain latent ophthalmoscopically.

A 76 year old patient was admitted in the hospital because of severe hypertension and marked cerebral sclerosis. He showed a moderate retinitis with thin arteries and normal papilla. Microscopically only the right side could be examined.

In the retina some albuminuric exudates were found in the outer layers, in the inner layer here and there longitudinal cavities and in some places lacunae of the layer of ganglion cells. The small vessels of the retina were partially thickened and closed. In the optic nerve there existed a continuous fairly widespread degeneration, which was situated in the forward region more in the peripheral part of the cross section and further backward more in the central part.

In contrast to the severe changes in the retina described above, in this case ophthalmoscopically there was only a mild retinitis. In spite of this there was a most definite degeneration of the optic nerve due to the destruction of large regions of ganglion cells in all probability caused by the diseased arterioles in the retina. The result insofar as the clinical examination is concerned is that we cannot foretell ophthalmoscopically in how far a process in the retina leads to destruction of the innermost layer and therewith to an ascending atropic process in the optic nerve. But if one finds in such a mild case of retinitis obviously bad functioning of the eye, one will have to remember such cases as this one. It is also doubtless of clinical interest, that very definite atrophic processes go on in the optic nerve which do not manifest themselves by pallor in the optic disc.

b) *Independent Processes in the Optic Nerve*

We have seen in the last paragraph that not seldom one can witness a certain incongruity between the severity of affections in the retina and processes in the optic nerve. We sometimes had the impression that the degenerative optic nerve process was of a secondary ascending nature. Now there are observations of mild retinitis without an extensive disease of the retinal arterioles, but in spite of this there is still a definite atrophic process traceable throughout the whole optic nerve.

In such cases one has to ask oneself whether the process of degeneration in the optic nerve is not independent of the affection in the retina and whether it does not continue on both sides. There was support for such a belief in several of my observations in the fact that the intensity and extent of the degenerative processes in the optic nerve were strongest a few millimeters behind the bulbus and decreased toward the back as well as toward the front. Anyway such observations cannot be used for our question with absolute certainty. But a case which I shall describe later on, in which a retinitis was combined with an affection of the chiasma, is absolute proof for the above point of view.

Apart from the possibilities just mentioned I have during the course of my examinations observed various pictures the origin of which can be put down to hardly anything else but the conducting visual path. I mention them here in the form of several groups without intending to say something definite about their origin.

As the first group I want to discuss whether there are primary lesions in the optic nerve which might be connected with disease of the arteria centralis retinae, of course without the clinical picture of emboly.

I will show you photos of a 66 year old lady who had hypertension with some uremia and who died from an apoplectic stroke. There existed a mild retinitis and both optic discs ophthalmoscopically were slightly edematous, but not pale. Microscopically there was a definite arteriosclerosis to be seen in the inner layer accompanied with blocking of the minute vessels of the retina. Now the interesting point is that the right optic nerve showed marked degeneration, whereas the left one was not at all degenerated. I am inclining to the view that the degeneration in the right nerve is to be related to changes in vessels within the optic nerve. This point of view is favored by the following facts:

1. That in spite of similar processes in the retina the atrophy of the optic nerve is present only in one eye. (By the way, just on that eye in which ophthalmoscopically the involvement of vessels was smaller.)

2. Further, the fact that on the side where the optic nerve was atrophied, the intensity of the degeneration was maximal a few millimeters behind the bulbus and could only in a small part be traced to the optic disc.

3. Furthermore on the diseased side there existed some disease of the arteria centralis retinae, which had certainly not brought on the degeneration by itself, since neither clinically nor anatomically one could speak of consequences of a blockage of the central artery. However, this disease had spread to some branches in the optic nerve. At any case the severe endarteritis of the small vessels in the axial fasciculus stands probably in connection with the disease of the main vessels. It is unlikely that these small vessels should have caused the atrophic changes at the edge; but since in addition there were some vessels with fatty degeneration in their walls; it may be thought, considering the foregoing arguments, that the degenerative process had started in the optic nerve itself. However, one must admit that fatty degeneration of walls in smaller vessels in the septa and neuroglia by no means always cause such a degeneration in the nerve tissues.

Rapid Necrosis of Nerve Substance. — A rapid decay of visual functions, which is definitely to be related to processes in the optic nerve, occurs occasionally in malignant nephrosclerosis.

I was fortunate enough to observe and to examine anatomically some time ago a particular characteristic and explanatory case.

A 49 year old patient who had been leutically infected many years back was absolutely well till suddenly a mist developed in front of both eyes. A neuritis optica was diagnosed by an eye specialist. In the next two weeks some retinal hemorrhages and a commencing star figure were added to the findings. The vision decreased rapidly. Two weeks later he was practically blind. From the internal point of view there existed only a general hypertension. The lumbar pressure was not increased. The patient died four weeks after the beginning of the eye trouble from apoplexy.

In this case of typical malignant nephrosclerosis the fact to be noticed most was the sudden decay of the optic nerve tissue, which had spread to all the bundles of the optic nerve. The processes in

the retina were so minimal that there was no question of an ascending degeneration. On the other hand no trace of a descending degeneration from the chiasma downward could be found. It is very probable that the process started inside the optic nerve but we are unable to say yet how it came about.

The rather obvious conclusion that the cause of the degeneration was due to disturbances of circulation inside the optic nerve trunk, could not be confirmed by anatomical examination. However, on the other hand, it is to be assumed that in consequence of circulatory changes the drainage of lymph in the optic nerve was made more difficult and that the small amount of damming in the optic disc is to be connected with this, since a raised intracranial pressure was not found.

The ultimate reason of the rapid decay of the optic nerve tissue is therefore not clear. But it is to be assumed with fairly good reason that a serious disturbance of circulation in the smallest vessels of the optic nerve were causing these processes.

In the literature I found another observation made by Raubitschek (1914) which clinically resembles the above case in many respects, but which could not be examined anatomically. It is a case of severe prominent papillitis with functional disturbances in a patient who has hypertension. After two days this papillitis receded, with whitish discoloration of the optic disc and strongly constricted retinal vessels first in one, then in the other eye. Although the visual power improved, it was harmed permanently. At the time a local process in the optic nerve was suggested.

Glaucoma-like Cupping in Consequence of Retrobulbar Processes. — Among my anatomically examined cases there is a very interesting observation which is pertinent to the discussion of the question, how far a retrobulbar degeneration of the optic nerve can be in relation with a general hypertension and how far it is able to produce a glaucoma-like cupping of the papilla.

A 42 year old patient who had a secondary contracted kidney with an elevated blood pressure and finally extremely high blood nitrogen showed at the beginning of an observation period of a year and a half a physiological excavation of the optic cup. This apparently physiological excavation changed during this time into a glaucoma-like cupping one.

The microscopic examination showed almost no localized changes of the retina. On the papilla there was a deep excavation.

In the lamina cribrosa and behind it there were small cavernous areas of Schnabel and a localized atrophy which could be followed backwards only with difficulty. Single vessels were without changes. Now and then a few small vessels in the pia and in the septal tissue were thickened. Also on the right side there was a retrobulbar atrophic area.

The main process in both eyes was undoubtedly situated behind the globe and now it is a question whether such a retrobulbar process in the optic nerve can be correlated with a general hypertension.

It would be of great value if some conceptions in the literature could be verified. Morax says that the cavernous areas of Schnabel are due to a vascular obliteration of the small vessels. Scheerer talks of necrosis of the laminar vascular net, which would be possible in cases of arteriosclerosis. As interesting as these conceptions of Morax and of Scheerer are, there are no real facts but more theoretical ideas which led them to their convictions.

Also Siegert tends by an observation to the opinion that local circulatory disturbances in the anterior part of the optic nerve are of major importance.

In quite a different connection Koyanagi mentions that in cases of retinitis albuminurica several small branches of the vascular net of Zinn, which are of great importance for the nourishment of the papilla and the surrounding retina, nearly all showed hypertrophical or degenerative changes.

In my own observation it was of great interest that the glaucoma-like cupping developed under the control of the oculist. In all probability the localized degeneration at the anterior end of the optic nerve was caused by disturbances of the circulation, which were referable to the general hypertension. Yet the histological examination gave insufficient evidence for that.

Our observation is therefore inadequate to answer the former question, but on the other hand, it directs one's attention to a probable connection of a glaucoma-like cupping (especially in the case of glaucoma without rise of intraocular pressure) with degenerative retrolaminar processes which in their turn are probably due to organic or functional changes in the small vessels of the nerve.

c) *Descending Degeneration*

Another question is whether sclerotic small branches of the Carotids are able to produce a disturbance of the nourishment of

the intracranial opticus or the chiasma. At times this possibility has been pointed out, especially by Henschen. The often cited observation of Henschen is very complicated and aside from the fact that general hypertension of the patient is not mentioned, it cannot be brought as evidence for the question under discussion. Up to the present we have no certainty on this point.

In the following I will refer to clinical and anatomical observations which concern chiasma and which make it more than probable that degenerations in hypertensive patients can start in the chiasma.

First, a very interesting case of a 26 year old patient who was observed during three years and could be anatomically examined. She showed in 1936 a typical retinitis albuminurica, a peripapillary edema, thin retinal arteries, high general hypertension, albumin in the urine, no uremia, normal vision.

A year later there was no change of the fundus, but a decrease of the formerly normal vision to fingers at 1 meter and there was now a slight uremia (79.3 mg. per cent).

Another year later after an intensive internal treatment the edema of the fundus had disappeared, but there still were exudates and a star figure in the macula. Vision right 5/50, left 5/30. Now one could distinguish for certain a bitemporal hemianopic scotoma.

Another year later the patient was in a very serious general state. Uremia 330 mg. per cent, a condition of the eyes hardly changed.

The histological examination of the eyes showed a typical retinitis albuminurica: In the optic nerve there was (1) a slight degeneration of some bundles near the margin of the cross section, possibly caused by the retinitis, (2) on both sides a small part of total degeneration, with great probability caused by the chiasmatic process, (3) a disease of small vessels, which can be seldom demonstrated in such a clear way.

This observation has a special interest for us, because the bitemporal hemianopic scotoma, clinically observed, proves that there can exist a retinitis albuminurica and independent of it a process in the optic pathway. In which way this process in the chiasma developed, can not be stated because the chiasma itself could not be examined.

That separate changes in the chiasma can be found histologically was briefly published previously. In two cases of papil-

loretinitis albuminurica, a little cord-like degeneration was demonstrable in the chiasma by scarlet red coloration and this degeneration could not be followed further on towards the nerve and the retina. In these 2 cases it was probable, according to the general symptoms as well as to the prominence of the disc, that the intracranial pressure was elevated. Therefore, besides the circulatory disturbance induced by the small arteries one must think also of the possibility, that the elevated intracranial tension had influenced the chiasma.

In my opinion the elevated intracranial pressure as a pathogenetic factor has to be taken into consideration, because I saw a similar change, limited to the chiasma in a case of early choking from tumor.

In any case the conception seems to be justified, that, on the basis of general hypertension now and then changes in the chiasma occur which can lead to a descendent degeneration of the optic nerve.

CONCLUSIONS

In the previous paper we spoke about the changes in the optic nerve and chiasma in patients with high blood pressure. We did not detail processes at the papilla of which a lot could be said.

The pathological processes in the *trunk of the optic nerve* and on the *chiasma* in cases of general hypertension (mostly in cases of malignant hypertension) are more frequent and more variable than one believes generally and they warrant more attention.

Whether the alterations in the connective tissue as lymphocytic infiltrations around the central artery and in the septa (?) or peculiar proliferations of the connective tissue in the axial fasciculus are intimately connected with the general hypertension, this question must be left open; they were mentioned, because I saw them several times in different degrees and because they demonstrate that behind the globe things are happening which we cannot recognize clinically.

It is of more importance, that degenerative processes in the optic nerve can also occur latent (without ophthalmoscopical signs). It is probable that they cause sometimes, according to their situation and extension, disturbances of the visual functions. Furthermore it is remarkable that by no means the retinal lesions of especially long duration produce an ascendant degeneration, but on the contrary just those forms which show clinically and anatomically few typical retinal spots. Instead of this the small retinal

vessels show a high grade arteriolosclerosis with obliteration of their lumen and lead in this way to severe disturbances at the inner layer with the consequences in the nerve fibres.

Moreover it can be regarded as certain that processes in the optic nerve and in the chiasma may occur which are not dependent on the retinitis albuminurica. Retinitis and processes of the peripheric pathway can occur concurrently as is evident especially well in a long observed case of retinitis with bitemporal hemianopic scotoma.

It seems that such independent diseases of the peripheric pathways can occur with different clinical aspects as circulatory disturbances, as glaucoma-like cupping, and moreover in the form of a rapid decrease of the vision combined with neuritic atrophy. For these several possibilities we showed observations, most of which could be studied clinically and anatomically. It is deplorable that the manner of their development is often still undiscovered. It is quite possible that the changes of the small vessels are of great importance and some of our statements can be employed in this sense, but an absolute proof in this direction has not heretofore been obtained. It is to be hoped that further systematical researches and favorable cases may lead to a more certain comprehension.

ACUTE HYDROPS OF THE CORNEA COMPLICATING KERATOCONUS*

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ALTHOUGH the literature on keratoconus is voluminous, reports of acute ectasia occurring in the course of the disease are relatively rare. Such reports as are available in the literature are recorded under the title of acute ectasia although ectasia is not always marked nor is it always present. The bulging of the cornea, its increase in depth and the opacity of the stroma are all due to its invasion and water-logging by the aqueous humor and for this reason we feel that the term "Acute Hydrops of the Cornea" is more definitely descriptive of this complication of keratoconus. The opportunity to study such a case and to record the progressive steps in the condition by photography and drawings impel us to publish this report in order to provide some encouragement for patients and oculists who may at some future date face this alarming complication.

Duke-Elder states that this condition "is the result of a rupture of Descemet's membrane and its endothelial lining, which is immediately followed by an opacification and swelling of the corneal tissues due to the entrance of aqueous into the stroma. As soon as the endothelium regenerates, the cornea regains its original appearance although some new opacity may remain. The ectatic portion usually is confined to the central part of the cornea and rarely involves more than half its area, the apex of the curve being eccentrically placed somewhat downwards and nasally." Applebaum, in an extensive review of the subject of keratoconus, offers

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a similar explanation as the cause for the complication of ectasia. Neither speculate as to whether Descemet's membrane ruptures are due to increased pressure in an abnormal eye, or whether the tissue is diseased or sensitized due to endotoxins.

Berner recently reported such a case and reviewed the literature very completely, his bibliography containing six previous reports by Terrien, Rabinovitch, Cooke, Reid, Rollet, and Goulden. The end results of some of these were not as satisfactory as that experienced in his own patient on whom paracentesis was done daily for four days, accompanied by the application of a 1 per cent solution of formalin to the apex of the ectasia in order to produce a resistant scar.

Terrien in 1906 protected the cornea in his patient, a female, aged 43, by a median tarsorrhaphy, noting regression of the staphyloma as well as the corneal opacity. Visual acuity returned to 6/60, the same as recorded before the hydropic invasion of the cornea. His impression of the production of the complication was the rupture of the endothelium and Descemet's membrane followed by an imbibition of aqueous by the corneal stroma. His description of the clinical appearance of the cornea at the onset of the condition was exactly that observed in our patient.

Rabinovitch in 1908 described an acute transitory ectasia complicating keratoconus in which recovery ensued, but the treatment was not described.

Cooke presented before the Royal Ophthalmic Society in 1911, a man, aged 28, who had suffered this complication of conical cornea. An elliptical portion of cornea, 3 mm. by 6 mm. was excised from the outer portion of the vertical diameter of the ectasia and healing progressed satisfactorily. Visual acuity improved from 6/60 to 6/18.

Reid reported a similar case before the same Society in 1924. The cornea of his patient, a youth of 18, was globate rather than ectatic. Treatment consisted of an inverted T shaped incision at the lower part of the cornea with drainage of the aqueous; the corneal opacity steadily decreased in area and vision improved from 6/60 to 6/18.

Rollet in 1929 gave the first complete report with slit lamp findings, showing that the thickening of the cornea took place in the substantia propria. He performed a sclerectomy on his patient, a girl of 18 years. The corneal staphyloma was reduced and clearing of the cornea took place.

Goulden's case was one of keratoconus accompanied by Descemetocele and does not seem to meet the requirements for inclusion in this series of cases of acute hydrops.

A search of the literature, in addition to the above reports gathered together by Berner, yielded but two other case reports. Butler in 1932 reported a case of acute conical cornea with thickening due to hydropic invasion which was accompanied by the curious formation of a bleb on the posterior corneal surface. The hydropic opacity, bleb and thickening of the cornea all disappeared after eight months, but vision was reduced to perception of light. Apparently the visual loss was due to some cause other than corneal opacity. No treatment had been given during the course of the acute stage.

Rugg-Gunn presented before the Royal Society of Medicine in 1935 a patient of Doctor Stutterheim of Johannesburg, who had keratoconus with a visual acuity of OD 4/60, OS 3/60. Several years later he suffered acute transitory opacity of both corneae. Under treatment the bulging had subsided, but an opacity persisted. The treatment was not described, but apparently was medical only. The patient was fitted comfortably with contact glasses with which he obtained a visual acuity of OD 6/12; OS 6/18.

Berner's case reported in 1936, was a girl, aged 14 years, who suddenly developed acute hydrops and true ectasia, following a game of basketball in which there had been no direct trauma to the eye. Paracentesis performed for four consecutive days was effective in reducing the ectasia. Clearing of the cornea resulted, and with a contact glass the final visual acuity of 6/9 was obtained.

Case Report. — B.A., a white male, was first seen when 26 years of age by one of us. (DBK), June 13, 1927, when he was a student in New York City. His chief complaint was failing vision for the past four years. He had been wearing O.D. +1.00, Sph. —2.75, Cyl. Ax. 75, and O.S. —.75, Sph. —1.75, Cyl. Ax. 15. He accepted O.D. +1.25, Sph. —5.50, Cyl. Ax. 75 = 20/20, and O.S. —.75, Sph. +3.00, Cyl. Ax. 10 = 20/20. Because of the irregular astigmatism which was detected with the aid of the keratometer, and because of the increasing astigmatism, the diagnosis of early keratoconus was made.

July 14, 1928, a progressive change was again noted. He accepted O.D. +2.50, Sph. —6.50, Cyl. Ax. 70 = 20/40+, and O.S. —1.00, Sph. +3.50, Cyl. Ax. 10 = 20/30. In 1930, Zeiss contact

glasses of regular curvature were tried with O.D. a 7.1 corneal curvature combined with —4.50 gave 20/20 —, while with O.S. an 8.1 corneal curvature, and no accessory lens, gave 20/20 —. No record of the scleral curve was made. He accepted O.D. +4.50, Sph. —8.00, Cyl. Ax. 85 = 20/30+, and O.S. +4.00, Sph. —8.00, Cyl. Ax. 105 = 20/30+. At this time, the diagnosis of keratoconus was easily confirmed by the clinical appearance of the conical cornea, and by the pulsation of the apex of the cone as detected with the keratometer. The regular Zeiss ground contact glasses were not tolerated, but Muller blown glasses were found after numerous trials from a large stock of lenses supplied by a New York firm which were suitable for each eye, and which were worn with satisfaction for fairly long periods of time. The patient became very adept in handling them.

In April, 1933, he wrote as follows: "No matter in what work I may engage, I am dependent on contact glasses as I have no working vision without them. I wear a glass in only one eye at a time, alternating eyes each day. I wear the glass continually for eight hours to nine hours without any discomfort, and then after a rest period of about two hours while at dinner, I can use the glass again for two or three hours in the evening, or if the occasion demands, I can wear the glass either in one eye or the other for a period of fifteen to sixteen hours."

Three complete investigations of the general condition were made in the effort to discover the cause of the development of the keratoconus. The results were inconclusive, and may be summarized as follows: There is no history of relevant eye disease in his family. The patient never ate vegetables or fruit as a child, and eats very little of them now. He has always eaten sufficient quantities of meat, milk and eggs. He had pneumonia without complications at the age of three. He had frequent attacks of epistaxis, but cauterization stopped them. He had measles and tonsillectomy at 28 years of age. Physical examination revealed that he was a well-developed and nourished male of stocky, short frame, with profuse hair on chest, abdomen and extremities. The sexual development was normal, with no evidence of glandular deficiency, except in his short stature and in a basal metabolism rate which was usually minus, averaging minus 18%. He had had much dental caries and many fillings. Blood pressure 114/72. Laboratory data: Hemoglobin 95%, Red Blood Corpuscles 5,620,000, White Blood Cells 4,400. Polymorphonuclears 72%, Smear normal. Urine:

Specific Gravity 1008, Acid, Albumen none, Glucose none, Microscopic negative, Phthalein 60%. Concentration Test: Specific Gravity (1) 1.018, (2) 1.020, (3) 1.021, (4) 1.022, (5) 1.24. Glucose tolerance, Blood Sugar (1) 100.1 (2) 150.1, (3) 202, (4) 155, (5) 142. Blood Wasserman negative in both antigens. Kahn negative. Blood Calcium 10.9 Mg.%. Blood Phosphorus 4.3 Mg.%. Blood Chlorides 6.44 grams per liter. Serum Protein 7.25%. Blood Creatinine 1.5 Mg.%. Blood Cholesterol .238%, Blood Urea .29%. Uric Acid 3.95 Mg. per 100 cc. Blood CO2 59.8 vols. %. Basal Metabolism minus 18%. Stereoroentgenograms of the chest were normal. There was no evidence of persistent thymus.

Summary to 1936: A case of keratoconus which was studied for a clue as to etiology and treatment. All tests were within normal limits. A history suggesting vitamin deficiency was obtained. On this basis, a diet high in vitamin content was recommended for trial.

In 1933, the patient developed the first deep opacity in his left cornea. Between 1933 and 1936, several other opacities formed in each cornea. These may well be interpreted as minor ruptures of the endothelium and of Descemet's membrane with rapid healing and with relatively minor symptoms and signs of change in the course of a case of progressive keratoconus. The use of the contact glasses probably did not produce the ruptures by trauma, as this patient was very careful in their use.

During the summer of 1936, this patient was in Memphis and he wore his contact glasses every day. The right one had been worn on the day preceding the episode to be reported for a total of eight hours. At 4 o'clock on the morning of August 22, he was awakened by a slight pain in the right eye. Immediate inspection in a mirror by him showed an opacity involving practically the entire right cornea. Vision was reduced to hand movement. The feeling of discomfort in the eye persisted for four hours. He had been swimming the previous evening, but there had been no diving or any strenuous exertion in the water.

The episode occurring on a week-end, it was thirty-eight hours later before he was seen by one of us (ROR). The appearance of the cornea was that of an acute ectasia as illustrated by Munson's sign in the photograph. Munson's sign has been admirably illustrated and described in Applebaum's review, and is a definite aid in the diagnosis of keratoconus. With the examiner in front of the patient, and with the upper lids raised, the patient is directed to

FIG. 1 — (Rychener and Kirby) — Munson's sign illustrating acute ectasia complicating keratoconus in right eye. A contact lens obscures keratoconus left eye.

look downward until the free margin of the lower lid bisects the cornea horizontally. The angle thus formed in the margin of the lid becomes a measure of the angle of the apex of the conical cornea. This ectasia was gray, globate and involved the central eight millimeters. The eye-ball showed a faint ciliary flush, but nothing in proportion to the corneal changes. These consisted of a discoid opacity with sharp outlines involving all layers of the cornea, the total depth of which was one and a half to two times that of the left eye. This could be differentiated into a central anterior striate opacity similar to that in the apex of the left cornea, a deeper translucent grayness of the substantia propria, which accounted for the increased corneal depth, and posterior to this, some broad vertical and oblique lines of steely, gunmetal relucency which were interpreted as folds or breaks in Descemet's membrane. A light pressure bandage was applied, and a drop of one-quarter per cent solution of eserine salicylate was instilled every four hours.

Reasoning that this acute phase was due to rupture of Descemet's membrane and the endothelium with edema of the stroma, and that lowering the tension would aid in regeneration of the endothelium over the breaks in Descemet's membrane, paracentesis was done, and repeated twice daily, by inserting a spatula through the conjunctival tunnel into the anterior chamber. The ectasia, which measured by the exophthalmometer was one-half to one

FIG. 2 — (Rychener and Kirby) — Front view of right eye of male patient with acute hydrops of cornea complicating keratoconus.

FIG. 3 — (Rychener and Kirby) — Right eye of same patient (side view). Note ectasia of superior temporal portion of cornea.

millimeter in advance of the apex of the left cornea, receded a full millimeter after each paracentesis, but made no permanent regression. After a week of such management the eye developed ciliary injection and became painful, whereupon atropin was prescribed with a prompt relief of symptoms. At this time the evacuated aqueous contained some fibrinous threads.

Drainage of the anterior chamber continued until September 7th, when it became necessary for the patient to go to his home

in the West because of the illness of his father. He was seen there by several consultants, and the paracentesis having become quite a painful procedure, he decided to dispense with the treatment. It had been done a total of twenty-six times.

Upon his return to Memphis a week later no change in the corneal opacity could be detected. Treatment with atropin, dionin, hot applications and light pressure bandage was continued, but no further surgical measure was attempted or given serious consideration. General physical examination disclosed no abnormality save a lowered metabolism for which during the previous two years he had continued to take thyroid extract in the dosage of one grain per day. Dental examination disclosed much pathology including pyorrhea, Vincent's infection, devitalized and abscessed teeth. It was deemed advisable by his dentist, because of the severity of the Vincent's infection, to give considerable sodium cacodylate intravenously before attempting extraction.

On September 22nd, having finished his work in Memphis, the patient left for New York City to return to Dr. Kirby's care.

Upon the admission to the Institute of Ophthalmology, the diagnosis of kerato-iritis complicating rupture of Descemet's membrane and ectasia of the cornea was confirmed. The water-logging of the corneal stroma and the inflammatory reaction because of focal infection in the mouth seemed to be the most important features. Non-specific protein therapy in the form of intramuscular injections of boiled milk was given. The infected teeth were carefully and gradually removed to avoid any focal reaction in the eye. Local treatment consisted in the use of atropin and hot compresses. The grayish white opacity and the increase of the cornea to two or three times its average thickness did not change much for a long time. Apparently it takes quite a while for a major breach of the endothelium and of Descemet's to heal. Perhaps the presence of focal infection sensitizing the tissues and producing kerato-iritis delayed the healing of the rupture.

He remained in New York for about a month and returned to his home where the treatment consisted of the application of hot packs to the eye for ten to fifteen minutes twice daily. This was maintained with diminishing regularity for a year. After that they were used two or three times a week for another six months. One drop of 10 per cent solution of dionin was instilled each night for a year, then replaced by a drop of glycerin in boric acid solution, and these were later alternated on successive months.

The patient was again in Memphis during the summer of 1938, and was repeatedly studied from July 8th to September 3rd. Remarkable clearing of the cornea had taken place. The remaining opacity consisted only of the subepithelial central opacity of the apex present at the first examination, and some translucent posterior lines in Descemet's membrane consisting of a vertical spindle with horizontal and apical branchings. The cone had entirely disappeared and an actual flattening of the cornea had developed. The diameter of the cornea had not changed, but the radius of curvature had definitely been altered. This was evidenced by the shift in the refraction.

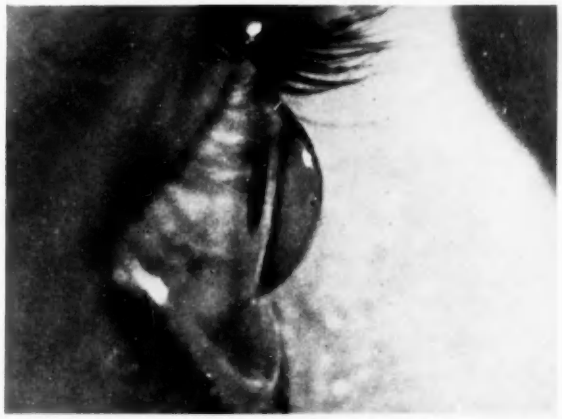
FIG. 4 — (Rychener and Kirby) — Clearing of cornea after two years. Visual acuity with correction, 6/9 and Jaeger 1.

O.D. 6/60 and Jaeger 16 improved with a +12.00, Sph. +5.00, Cyl. Ax. 120 to 6/9+; the addition of +1.00 Sph. gave Jaeger 1, easily.

O.S. with contact glasses 6/30 and Jaeger 1 improved with —1.00, Sph. +3.00, Cyl. Ax. 105 to 6/9 partly.

The correction for the right eye was prescribed in ordinary lenses, and a letter written November 3, 1938, stated, "I am getting along fine, and it is a wonderful thing to be no longer dependent on a contact glass for reading and ordinary working vision." The change in the refraction was quite remarkable.

On the night of November 11, 1938, this patient experienced in his left eye the same interesting phenomenon as had occurred two



FIGS. 5 AND 5A — (Rychener and Kirby) — (5) Side view of both eyes of same patient two years after acute hydrops in right eye. The right cornea is thickened but normal in contour. (5A) Keratoconus persists in left eye.

years previously in his right, namely, bulging and opacification of the left cornea. He did not have a paracentesis done at this time preferring to carry on some experimental work on his own initiative. Treatment has consisted of hot packs applied regularly twice daily, and after a month the daily instillation of one drop of 5 per cent solution of dionin. Vision was reduced to hand movements at a few inches, but after three months clearing began with visual improvement. The acute bulging of the cornea had also decreased at that time. No iritis occurred at any time, and it is possible that

the repeated paracenteses in the presence of focal infection were responsible for this complication in the right eye.

He has continued his work daily since the acute hydrops in his left eye, driving his car to and from his place of business, and maintaining his usual reading and other close application of his eyes. All of his work has been done with the ordinary lens for his right eye (+12.00, Sph. +5.00, Cyl. Ax. 120). This patient was again examined by one of us (DBK) on May 17, 1939, with the following findings:

"Brows and lids negative. The palpebral fissures measure O.D. 9 mm., O.S. 8 mm. There is no congestion of either eye. The cornea in each eye measures 12.5 mm. in diameter. The right cornea is clear except for a vertical linear opacity 4x1 mm. The left cornea has a 6 mm. disc-like central opacity. With the slit lamp microscope: O.D. There is an opacity in the substance of the cornea. At the upper and lower poles of this opacity are 3 or 4 radiating lines formed by folds 1 mm. in length in Descemet's membrane. There are a few deeply placed vessels.

"O.S. There is a dull, translucent, nebulous opacity of moderate density. The optic section of the cornea shows a normal thickness at the periphery, increased thickness at the edge of the opacity, and thinning at the center. There are a few deeply placed vessels. The anterior chambers are of average depth. The irides, pupils and lenses appear normal. Tension normal to fingers. Exophthalmometer same in both eyes. The prospect is that there will be further clearing of the left cornea."

EXPERIMENTAL WORK

The flattening of the cornea and the change in the radius of curvature as indicated by the remarkable shift in refraction following this acute hydropic change caused one of us (DBK) to raise the question whether or not this complication was a conservative one. Visual improvement has taken place in most of the cases reported despite a variance of methods of treatment. If rupture of the endothelium and Descemet's membrane resulted in a beneficial change in such eyes, one of us (ROR) wondered why would it not be advisable to deliberately induce such a hydropic change when the keratoconus is of very high degree. Such eyes with extreme keratoconus may be lost from corneal ulceration, perforation and panophthalmitis.

With this theory in mind, some experimental work was undertaken by one of us (ROR), in a series of twenty-four rabbits, in

which efforts were made to reproduce acute hydrops of the cornea by lacerating the endothelium and Descemet's membrane from the direction of the anterior chamber.

Leber in 1873 produced edema of the cornea by denudation of the endothelium which was followed by opacification and swelling of the cornea. He and others (Panas, Rampoldi, Mellinger, Barri, Elschnig and Juler) demonstrated that this could be brought about not only by instrumentation but also by the introduction of chemicals into the anterior chamber, that the hydropic condition persisted until the endothelium regenerated over the injured area, and that occasionally an opacity remained permanently.

Such a condition of acute corneal edema was reproduced without great difficulty as revealed by the accompanying photographs after a certain technical procedure was determined by trial of various methods. The insertion of a knife needle with a curved tip through the cornea near the limbus, turning the knife edge forward and making several incisions through the endothelium and Descemet's membrane in the central area resulted in immediate opacification of the cornea of various degrees. However, this method also traumatized the iris and lens in a number of cases so that it was discarded for another technique, which has proved to be quite satisfactory.

General anesthesia was produced by the intraperitoneal injection of a solution made up as follows:

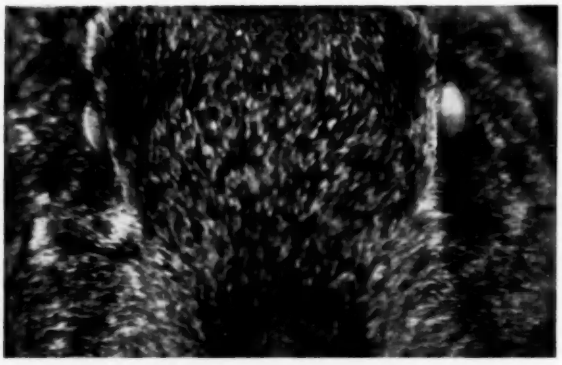
Sodium barbital	5
Barbital	20
Urethane	100
Aq dest.	140

1.5 cc. of this solution per kilogram of rabbit weight was the dosage used, affording satisfactory and complete relaxation which persisted from twelve to twenty-four hours.

A small keratome incision directed centrally was made in the cornea 4 millimeters from the limbus. The chamber emptied practically entirely, and a curved cystotome or a foreign body spud with a sharp right-angled point was then inserted on the flat, the tip passed beyond the center of the cornea, the sharp edge turned forward and the posterior corneal layers lacerated by drawing the instrument toward the point of entrance. More than one such laceration could be made, if desired, before withdrawal of the instrument. It was found that the Barkan goniotomy knife made an ideal instrument for this purpose. Immediate opacification took

place in a small degree, and increased in some cases to involvement of the entire cornea within forty-eight hours. Clearing began after the height of aqueous invasion was reached and was complete within two weeks to two months. Deep striate opacities of the cornea remained permanently, however, and corresponded to those deep opacities which have persisted in cases of keratoconus complicated by acute hydrops.

Under the technique finally adopted, it was possible without complications to produce acute hydrops of the cornea in twenty-eight consecutive eyes. It was surprising to find it possible to insert the instruments mentioned into the anterior chamber without trauma to the lens, even though the chamber seemed to be optically empty. However, McMillan in his work on filling the anterior chamber with air after trephine operation had a similar experience.



FIGS. 6 AND 6A — (Rychener and Kirby) — (6) Front and (6A) side views of experimental hydrops of cornea induced in rabbit twenty-four hours posterior keratotomy.

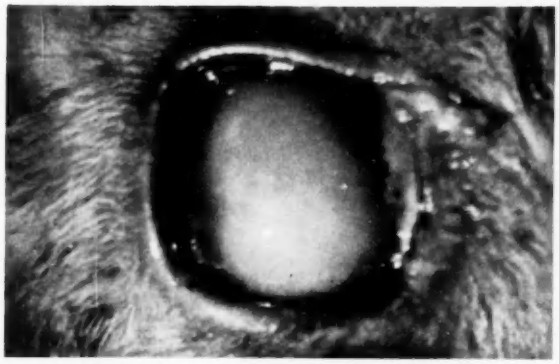


FIG. 7 — (Rychener and Kirby) — High power of left eye of same rabbit. Increased density and ectasia of cornea in superior portion.

FIG. 8 — (Rychener and Kirby) — Clearing of cornea one month after acute hydrops induced by posterior keratotomy.

EXPERIMENTAL PATHOLOGY

The eyes of several rabbits were removed forty-eight hours after instrumentation, fixed in formalin, 5%, and sent to Doctor Georgiana Dvorak-Theobald, who kindly prepared and sectioned them.

Sections demonstrated that an acute edema of the cornea had taken place, the cornea being swollen two to two and one-half times its normal thickness because of an imbibition of fluid in the stroma. The sections were cut so as to demonstrate the keratome incision as well as the lacerations in the endothelium and Des-

cemet's membrane. These latter were somewhat magnified because the sections passed through them diagonally, but demonstrate forcefully the elasticity of Descemet's membrane and the extent to which it retracts and rolls in upon itself when it is cut or broken.

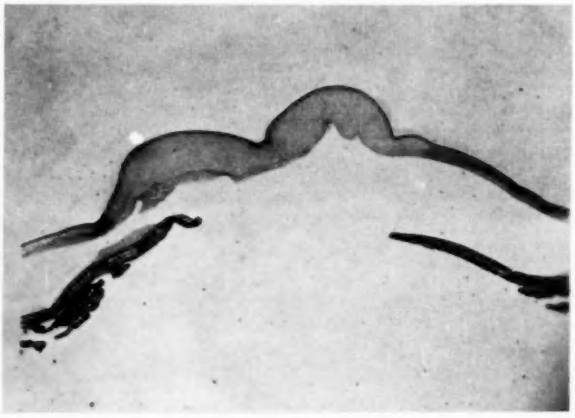


FIG. 9 — (Rychener and Kirby) — Acute hydrops of cornea in rabbit's eye, twenty-four hours after posterior keratotomy. Descemet's membrane is retracted and curled upon itself. A fibrinous exudate is present on the posterior surface of the cornea and the anterior surface of the iris.

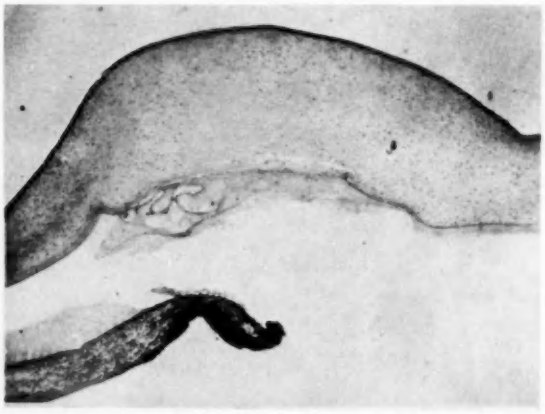


FIG. 10 — (Rychener and Kirby) — High power of same section as Figure 9. Corneal stroma is three times the normal thickness, with separation of the lamellae. Marked retraction of Descemet's membrane.

Sections from similar eyes enucleated a month following instrumentation showed complete regression of the hydropic stage. The cornea had returned to its normal thickness following regeneration of an impervious endothelium and only a mild fibrosis marked the area of trauma.

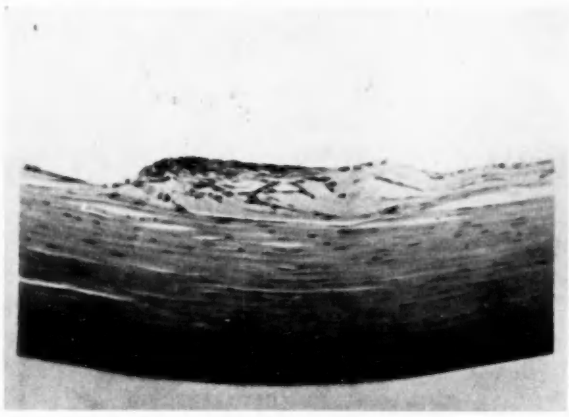


FIG. 11 — (Rychener and Kirby) — High power of section of cornea from rabbit's eye one month following posterior keratotomy and after clearing of the cornea had occurred. The endothelium has regenerated and a mild fibrosis covers the area of trauma. The cornea has resumed its normal thickness.

Sections from rabbit eyes enucleated twenty-four hours following the induction of acute hydrops of the cornea by posterior keratotomy were also examined by Doctor John S. McGavic who found as follows: "The corneal stroma varies from normal thickness to an increase of three times over the normal in the central region. Here the lamellae are widely separated and partially split, fine trabeculae connecting the parts of the lamellae. Descemet's membrane has been ruptured in one to three places in different sections beneath the area of the hydropic stroma described. Descemet's membrane takes a deeper stain than usual and is about twice as thick as normal. The endothelium has a ragged vacuolated appearance. In some sections there is a fibrinous exudate in the anterior chamber just back of the injury to Descemet's membrane. The angle of the chamber and the iris are normal. There is a cellular exudate on the surface of the ciliary body.

The second eye shows the same stromal changes and injury to Descemet's membrane. However, Descemet's membrane is of normal thickness throughout. The endothelium shows the same

change and there is a similar fibrinous exudate behind the injured area. In this eye there is no cellular reaction on the surface of the ciliary body."

From the work reported above it would seem that another surgical approach may be made in the treatment of keratoconus of a high degree where ulceration is impending. It is quite true that experimental keratoconus was not produced and reduced by this experiment. However, clearing of the cornea with actual flattening has occurred in the case of acute hydrops complicating keratoconus which are recorded in the literature, and it seems reasonable to advocate the deliberate production of acute hydrops in some cases of advanced keratoconus. Certainly such a procedure need not be more difficult than the excision of an elliptical portion of the cornea, nor produce more actual trauma to the eye than perforation of the cornea by actual cautery, and in the light of experimental results it seems more logical.

SUMMARY AND CONCLUSIONS

1. An additional case of acute hydrops of the cornea complicating keratoconus is added to the eight cases of a similar condition previously reported in the medical literature.

2. In spite of the varied forms of treatment nine of the ten eyes reported to date have maintained or actually improved the visual acuity previously recorded. In the single eye in which blindness occurred the cornea had cleared and the visual loss was due to some cause other than the complicating corneal edema.

3. Reduction of corneal ectasia and clearing of the corneal edema occurred uniformly in all cases regardless of the form of treatment used. Procedures such as tarsorraphy, sclerectomy, keratectomy and keratotomy were invoked to prevent a spontaneous rupture of the whole thickness of the cornea, a condition which has not as yet been recorded.

4. Experimental hydrops of the cornea has been produced in rabbits and spontaneous clearing of the cornea took place after periods varying from two weeks to two months. Striate opacities corresponding to the lacerations in Descemet's membrane persisted, and were similar to those described following resolution of the complication in human eyes with keratoconus.

5. Posterior keratotomy is offered as a rational procedure in eyes affected by keratoconus in which visual acuity is reduced to a very low degree or in which the protrusion of the cornea is of such high degree that exposure of the cornea and its subsequent complications endanger the integrity of the eyeball.

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DISCUSSION

DANIEL B. KIRBY, M.D., New York: I congratulate Doctor Rychener for his original research bearing on the subject. He has turned what would otherwise be a routine report of an interesting condition into a valuable contribution. It is true that he has not used the procedure of keratotomy in an extreme case of keratoconus but he has laid a good basis by his experimental work for his recommendation that it might be tried.

The occurrence of the acute hydrops in this patient's right eye naturally caused great fear in this patient and his fears were not at all relieved by his physicians or consultants despite the graceful outcome of the cases reported in the literature. All were extremely pessimistic about the prognosis and when with the healing of the rupture of Descemet's and of the endothelium, the cornea regained its transparency and resumed a more normal thickness and curvature with restoration of useful vision, the patient looked with equanimity on the similar event in the left eye.

The production of hydrops in this patient's eyes and in the experimental animals confirms the opinions concerning the production of corneal edema and even of some cases of bulbous keratitis by posterior corneal trauma during eye surgery.

I saw this particular patient, a white male, first in 1927. He complained of failing vision for four years. He had been wearing a correction for a moderate degree of mixed astigmatism in each eye. He accepted: O.D. +1.25, Sph. —5.50, Cyl. ax. 75 = 20/20 and O.S. —.75, Sph. +3.00, Cyl. ax. 10 = 20/20. Because of the irregular astigmatism and of the considerably increased astigmatism, we made the diagnosis of early keratoconus. In 1928, a progressive change was again noted. Zeiss contact glasses were tried but not tolerated. Muller blown contact glasses were selected from a large stock, two of which were found suitable for each eye and they were worn with comfort and excellent visual results. The patient was dependent on them for all of his work. He became very adept in their use, wearing them continually for 8 to 9 hours per day and on occasion alternating one eye and then the other for a period of 15 to 16 hours in one day. The diagnosis of keratoconus was easily made in 1928 by the clinical appearance and by the pulsation of the apex of the cornea.

We made three complete investigations of the general condition of this patient. The results were inconclusive but may be summarized as follows: No relevant family history. The patient never ate vegetables or fruit as a child and even now he must be forced to do so. He has lived chiefly on meat, milk, eggs, bread and butter. His nutrition appeared good. His frame was short and stocky and covered with profuse hair. He gave the appearance somewhat of pituitary dystrophy and his basal metabolism rate was minus 18 to minus 20. Elaborate blood and urine chemistry examinations did not reveal anything significant.

In 1933 he developed his first deep opacity in the left cornea. By 1936, several such opacities had formed in each cornea. These were regarded as minor ruptures of the endothelium and of Descemet's membrane. The use of the contact glasses may have had something to do with their development in this case, but they are such as are seen frequently in other cases of keratoconus where contact glasses are not used.

Doctor Rychener has told you of the acute episode in the development of hydrops of the right eye in 1936. I had the privilege of following the patient on his return to New York. His cornea was still water-logged and protruding. Iritis was also present. The inflammation had arisen as a result of focal infections in his mouth. A number of teeth were removed and non-specific protein therapy given. Atropine and hot compresses were employed. He remained in New York for a month and then returned to his home employing dionin, alternating with boric and glycerin, and hot compresses daily for a year and more. In 1938 and again in 1939 it was noted that remarkable clearing had taken place. The vision had improved so that at the present time (October, 1939) it is 20/30 with +11.00 Sph., +6.00 Cyl. ax. 125. The conus has disappeared and the thickness of the cornea appears uniform throughout. The change in refraction is remarkable and denotes a flattening of the curvature of the cornea. Some irregular astigmatism remains. There is good lustre and the cornea looks remarkably clear although on close examination fine deep radiating nebulous opacities are to be seen. The patient is well pleased with the return of useful vision in this right eye. The intraocular tension is 16 mm. R, 15 mm. L. Sch. The exph. reading was 16-15 Hertel.

The most interesting sequel of the development of the same phenomena of acute hydrops of the cornea of the left eye happened in November, 1938. He did not have any paracentesis this time and developed no inflammatory reaction. The opacity and bulging of the cornea were as great as that which had occurred in the right eye. The remarkable thing was the equanimity and hope with which the patient viewed this complication in his second eye. Now the left eye exhibits a flattened cornea with good lustre and thickness. There is irregular astigmatism of about seven or eight diopters. The vision is limited to 8/100 by a central nebulous opacity. We thought it would be necessary to do a corneal transplant for the restoration of central vision in the right eye but the natural recovery of transparency has rendered this unnecessary and the same may be true of the left eye after a greater lapse of time. The prospect is that there will be further clearing of the cornea. Conservative treatment is still indicated.

Knowing the literature on rupture of the endothelium and of Descemet's membrane and the results in this case, we may give a better prognosis when it happens in another patient and we will be on the look-out for a suitable desperate case of keratoconus in which posterior keratotomy may be useful.

HARRY S. GRADLE, M.D., Chicago: The new term introduced by the authors, "Acute Hydrops of the Cornea," is good and will undoubtedly find a permanent place in our nomenclature. Just how great is the rarity of this condition is very difficult to say for undoubtedly the majority of ophthalmologists of any great clinical experience have seen one or more cases, but failed to report them. I say this from personal experience for I can add two cases to those here reported.

This first was a school girl, 17 years of age whom I first saw in the middle of December of 1936. She gave the history of a bilateral keratoconus of some six months' duration. One week before I saw her, there had been a sudden loss of vision in the left eye, unaccompanied by pain. I found a low degree of keratoconus in the right eye. The left eye was lightly injected. At the apex of the cone was a definite keratectasia, nearly 4 millimeters in diameter. The entire central area of the cornea showed a milky-white opacity fairly regular in outline and involving the entire corneal thickness up to Bowmann's membrane. Details of the posterior corneal surface could not be seen through the opacity. The anterior chamber was very deep. On digital examination, the tension seemed to be normal or slightly minus.

An immediate iridencleisis was performed, followed by an uneventful recovery. Within three days the keratectasia had disappeared and the cornea began to flatten. One year later, the corneal opacity was visible only as a linear stripe, 3 by 1 millimeters in size. With a contact glass, 2/8 + 3.00, was nearly 0.6.

In June, 1938, the opacity was visible only biomicroscopically. With the same contact glass, which could be worn in either eye for as high as 14 hours, her vision was R. 0.8 and L. 0.6.

The second case was that of a young man, 23 years old, seen for the first time in May, 1938. He had evidently had a bilateral keratoconus for many years. One week before his visit, there had been a sudden loss of right vision unaccompanied by pain. In the right eye was seen a keratoconus with a 3 mm. keratocele bulging from the apex of the cone. Two-thirds of the entire cornea showed the typical milky-white opacity of aqueous infiltration, sufficiently dense to prevent a biomicroscopic view of the posterior corneal surface. An immediate iridectomy was performed with subsequent reduction of the keratocele within ten days. From then on, the cornea flattened perceptibly, but a large share of the opacity persisted. When last seen in July, 1939, the right vision, even with contact glass, was less than 0.1.

The left eye of this patient had a typical keratoconus with a mere suggestion of keratectasia when first seen on May 16, 1938. Eleven days later, that is to say, ten days after the right iridectomy had been performed, there occurred during the night a rupture of Decemet's membrane and an acute hydrops of the cornea. The extent of the opacity was not as great as in the right eye, but distinct vacuolization within the corneal stroma could be seen biomicroscopically. Owing to the presence of an acute conjunctivitis, operation was postponed for nearly three weeks and a pressure bandage was used during that time. This seemed to have no influence upon the keratectasia, so on June 16, 1938, an iridectomy was performed upon the left eye. Here too there occurred a marked flattening of the cornea with permanent reduction of the keratectasia. Thirteen months later, much of the opacity had cleared so that with a contact glass 2/8, 0.6 vision could be obtained in the left eye.

Thus to supplement Kirby and Rychener's report, are offered three eyes in two individuals, with keratoconus, keratectasia, rupture of Descemet's membrane and acute hydrops of the cornea. All three eyes were operated upon, one by iridencleisis and two by iridectomy. The most striking feature of the post-operative course was the marked flattening of the keratoconus and the failure to progress that is naturally expected in individuals of that age so afflicted. Undoubtedly, the acute hydrops resulted in the formation of connective tissue within the corneal stroma that prevented further bulging. Furthermore, the opacity in one of the eyes disappeared almost entirely; while in the other two it became permanent.

It is of course futile to speculate as to what might have happened had the condition been cared for by non-surgical means, but judging from the report of the authors as to the course of the left eye in their patient, it would appear that much time and energy can be saved by immediate surgical intervention. Apparently, iridectomy is simpler than multiple paracenteses, and iridencleisis appeared to be equally successful. The reason for any operative procedure is to affect an immediate reduction in intra-ocular tension which is undoubtedly too high for that particular eye, thus producing the keratectasia and eventually the rupture of Descemet's membrane by the simple mechanical process of stretching to the breaking point. A priori, a simple iridectomy is to be preferred to an iris inclusion operation because of the possible irritation of the subconjunctival iris by the contact glass that will eventually have to be fitted. However, practical experience has shown that this objection is more theoretical than real.

TRYGVE GUNDERSEN, M.D., Boston: The reason the corneal stroma swells or takes on fluid when directly exposed to the aqueous is not at all clear. It cannot be merely a matter of fluid being pressed into the tissues from the anterior chamber, because the corneal parenchyma will swell in the absence of any pressure. For instance, an excised cornea will swell in only a fluid bath. Also the corneal parenchyma's affinity for fluid seems even more remarkable since this affiinity is not directly attributable to osmosis. The cornea has been shown to take on fluid even when placed in a bath of 10 per cent sodium chloride, many times normal.

Leber's experiments also showed that the amount of swelling was practically the same if the immersion fluid were distilled water or 2 per cent sodium chloride. He contends that the swelling depends on the affinity of the fibrallae for fluid regardless of the osmotic pressure of the surrounding fluid. It is perfectly clear that the endothelium alone prevents this happening in the normal, living cornea. Descemet's membrane is doubtless of very little importance.

In the study of experimental keratoconus, Plaut, Elschning and Hess were able to produce marked swelling of the cornea by mechanical injury to the endothelium as well as by electric shocks, which did not disturb Descemet's membrane. It was found that it was unnecessary to disturb Descemet's membrane, even as von Hippel found it unnecessary to disturb it in his classical fluorescence experiment. Leber also found that excised Descemet's membrane offered no particular hindrance to the passage of fluids.

The fact that Descemet's membrane and its overlying endothelium ruptures in keratoconus suggests that there is an abnormality of this structure or that it is stretched in an abnormal manner. If the intraocular pressure in a normal eye is increased until the globe slowly ruptures, the first cracks do not appear on the posterior but on the anterior corneal surface. Ehlers has demonstrated this in the eyes of calves and I show you a lantern slide taken from his monograph. (Slide.) This represents the cracks in the surface of the cornea in the eyes of calves when the pressure is increased to about 3 atmospheres. One is also impressed by the elasticity of the normal Descemet's membrane, when one sees a descemetocele such as occurs in a case of corneal ulceration with sufficient loss of corneal substance to allow Descemet's membrane to bulge forward.

Finally I should like to draw attention to the similarity between Doctor Rychener's pathological specimens and the pathological specimens from patients with so-called sclerosis of the cornea reported by von Hippel, Meller, Fuchs and Gilbert. Sclerosis of the cornea is purely a clinical concept. It is an edematous thickening of the cornea which occurs without previous ulceration. It has been reported as occurring in cases of trachomatous pannus, sclerosing keratitis, old glaucoma, or where exudates have been layered on the posterior corneal surface for a long time.

(Slide.) In this lantern slide, taken from von Hippel's case, there is tremendous swelling of the corneal stroma with great increase in the corneal thickness. The lamellae are arranged in a peculiar wave-like fashion, and, as you see, the immediate cause of all this probably lies in the fact that the endothelium is completely absent.

(Slide.) This colored slide of the same case may show the pathological picture of corneal sclerosis a little more vividly.

T. L. Terry, M.D., Boston: In the past year two patients with conical cornea under my care developed a marked sudden increase in the height of the cones. Edema of the cornea was present and widespread, but it was not as extensive as that just reported and it was not the most important or the most obvious finding. One of these patients was treated with pressure applied by means of an elastic bandage and a remarkable reduction in the amount of the cone was produced in twenty-four hours, so great that surgical intervention, which had been considered, was not carried out.

The condition of the second patient can best be illustrated by a lantern slide of molds of the cornea made (1) preparatory to having a contact glass manufactured, (2) after the sudden increase of the cone, and (3) after treatment with pressure. For several days after the pressure was applied the cornea was moderately thin although the conicity of the cornea was fully reduced. Eventually, after the pressure had been continued for a long time, a period of ten weeks, scar formation was sufficient so that the normal curve of the cornea was restored. Some six months has intervened and this patient was able to resume her school work with practical vision, requiring no glasses for reading. The scar that resulted did not cover the entire pupillary area.

The term "acute hydrops of the cornea" is a very good one, but I do not believe it fits the particular type of cases I have mentioned as they showed much less edema of the cornea than the case of Doctors Kirby and Rychener.

Ramon Castroviejo, M.D., New York City: It is encouraging to see that these patients with very advanced keratoconus, just reported by Doctors Rychener and Kirby and Gradle and the one reported by Doctor Terry, have improved with such minor surgical procedures, but even so there are some cases of keratoconus which are so advanced they cannot be improved without surgical intervention.

Before the surgical procedures that were used did not lead to very good results. Nowadays, however, we have a method that offers the possibility of restoring very good practical vision to these people who before had very little hope of ever regaining useful eyesight again. At the Institute of Ophthalmology we have operated on eight cases for corneal transplantations, very advanced cases of keratoconus. We have taken those cases with very marked keratoconus, with scars in the apex of the corneas, cases that could not be improved before by wearing contact glasses, cases whose visions were very much diminished and they were impossible to improve by any means. The eight of them operated upon regained vision better than 20/58. Before the operation vision was less than 10/200.

This is only to encourage you that cases of very advanced keratoconus that could not be improved before can now be cured by transplantation. Keratoconus are the most favorable types of eyes for conical transplantation operations. They are more favorable than the interstitial. They are more favorable than the centralis corneal scars that follow the inflammatory process. They are the most favorable cases of all for the conical transplantation operation.

RALPH O. RYCHENER, M.D., in closing: The discussers have been very generous and very informative. To the foregoing have been added five eyes with the condition of acute hydrops of the cornea complicating keratoconus with four different methods of treatment, which added to those already described make nine different forms of treatment utilized for this condition and practically all of them have been successful.

What we need to do in the future for these cases is still of course a question that remains to be determined. Doctor Castroviejo's report is most encouraging. It would seem from his experience that keratoplasty is a method of choice for treatment of extreme keratoconus. However, it is not possible, because of economic reasons, for all patients to get into the hands of men as expert in keratoplasty as Doctor Castroviejo. Sometimes these people have to be cared for in some of the more outlying districts like our own and it may be that occasionally some simpler method will suffice.

TRANSPLANTATION OF CORNEA BY MEANS OF A MECHANICALLY OBTAINED BEVELED EDGE SEGMENT

MEYER WIENER, M.D., *and* BENNETT Y. ALVIS, M.D.*
ST. LOUIS

EVER SINCE transplantation of cornea has been seriously considered by eye surgeons, they have sought a means of obtaining a graft with a beveled edge, which might be obtained mechanically.

Several years ago we began experimenting with this end in view. The first idea was the use of a specially constructed trephine. We succeeded in this by using three tiny blades constructed with rounded, spade-like cutting edges, fitting into a cone shaped tunnel, and the whole mounted on a tripod, the feet of which rested on the corneal margin.

This worked very well theoretically and on stiff, resisting material such as leather. When it was tried on the cornea, the elasticity of the cornea prevented completion of the cutting of the segment and caused it to curl up and tear. This very elasticity which led to failure with the trephine is the specific property that makes possible the cutting of a beveled graft by means of a specially constructed punch.

An incision is made in the cornea at the upper corneal margin, large enough for the insertion of the punch blade. The punch is constructed so that the female blade is 6.5 mm. wide and 1.0 mm. thick. The upper surface of its bore measures 4.5 mm. in diameter. The under surface is wider, as the hole is cut on a bevel so as to make the upper cutting edge sharp.

The male blade is bullet nosed, its diameter 2.5 mm. from the tip, being the same as that of the hole in the female blade. When the sharp edge of the female blade, after being introduced into the anterior chamber, engages the under surface of the cornea,

* From the Department of Ophthalmology of the Washington University School of Medicine.

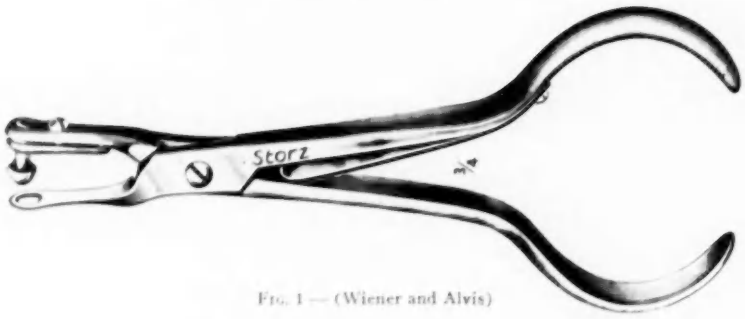


FIG. 1 — (Wiener and Alvis)

the tip of the male blade presses down, reverses the curvature and stretches the entire anterior corneal surface driving a wider portion of it into the punch.

By the time the epithelium reaches the cutting edge of the female blade it is stretched over the bullet nose of the male blade to such an extent that the segment punched out has a beveled edge, the epithelial surface of which is about 0.6 mm. wider than the endothelial surface. The graft is not injured either macroscopically or microscopically.

The operation is carried out in the following manner:

Local anesthesia is induced by the use of one-half per cent Pontocain instillations with retro-bulbar injection of two per cent Novocaine. Akinesia of the lids is done by the O'Brien or Van Lint method. A small amount of 2% Suprarenin Jel is then introduced into the cul-de-sac. This is done for a twofold purpose. It dilates the pupil ad maximum so that it will not contract on emptying the chamber. Since we have done this we have had little difficulty with prolapse of the iris, either at the limbal incision or at the site of the punch opening.

The lids are separated; with a double armed suture of the finest silk thread obtainable, armed with 10 mm. fully curved Kalt needles, a horizontal bite about 2 mm. wide is made in the cornea close to the upper limbus. Each needle then takes a small but firm vertical bite in the conjunctiva-sclera close to the limbus opposite the exit of the threads in the cornea. The loops are pulled aside. We are now ready for the modified Thomas suture. The center of the cornea is marked. Two fine lines are cut on the cornea through its center with the tip of a sharp scalpel at an angle of 45 degrees to the horizontal, making them perpendicular to one another. These are the markers for the stitch which is to hold the graft in place.

They are made visible by instilling a drop of Fluorescein and then washing out with sterile water or boric solution.

With the same suture material armed with a 10 mm. full curved Kalt needle, a bite is taken in the cornea, superficially entering at a point on the line toward the upper temporal corneal margin, about three mm. away from the margin. A second bite is taken opposite the first, emerging on the upper nasal line at a point corresponding to the entrance of the first bite on the opposite upper line. The needle then takes a small, superficial bite into the cornea near the lower temporal border at a point corresponding as nearly as possible to the first bite on the upper temporal line. The final bite enters the cornea below the entrance of the second bite, emerging on the lower nasal line also about three mm. away from the corneal margin.

The advantage of having the lines is that when the threads emerge on the lines, regardless of whether they have been equally placed, the crossing point of the threads is bound to be over the center of the cornea, thus holding the graft in position evenly. The loop suture is now carefully laid aside.

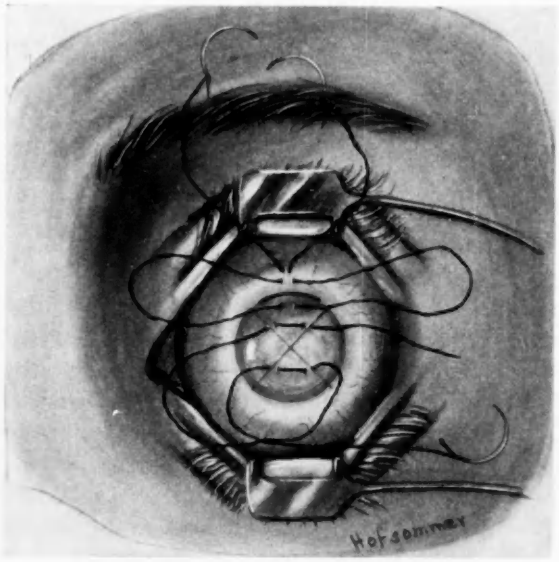


FIG. 2 — (Wiener and Alvis)

An incision is made in the cornea at the limbus, about eight mm. long, between the loops of the first thread with a short, narrow Graefe knife. The female blade of the punch is then introduced into the anterior chamber so that the middle will be opposite the center of the cornea. Holding it against the under surface of the cornea, the punch is slowly but firmly closed. The blades are then separated, and the female blade slowly withdrawn from the chamber, the while holding it close to the under surface of the cornea so that the excised portion will remain within the circle of the female blade. This is not so important in removing the first segment as it is in taking out the portion of the donor eye to be used as the graft.

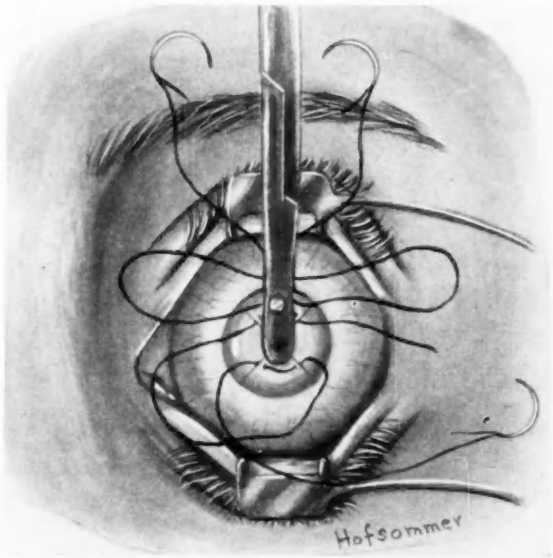


FIG. 3 — (Weiner and Alvis)

The upper suture is then tied, closing the limbal incision. The host is now ready to receive the graft from the donor. The segment is obtained in the same manner, except that after the eye has been enucleated and placed in a normal salt solution awaiting its use, we merely make the incision with the Graefe knife, insert the punch blade and remove the segment. The graft is carried from the donor eye on the punch blade to the cornea of the host where it is carefully slid off with the point of the Graefe knife into its position, whereupon the corneal cross stitch is carefully tied. Care

must be taken not to tie it too tight, since it tightens up considerably when the anterior chamber fills. If the stitch is snug on completion of the operation, it will be too tight later and press against the graft, distorting it and jeopardizing its vitality. The knot must not lie over the graft.

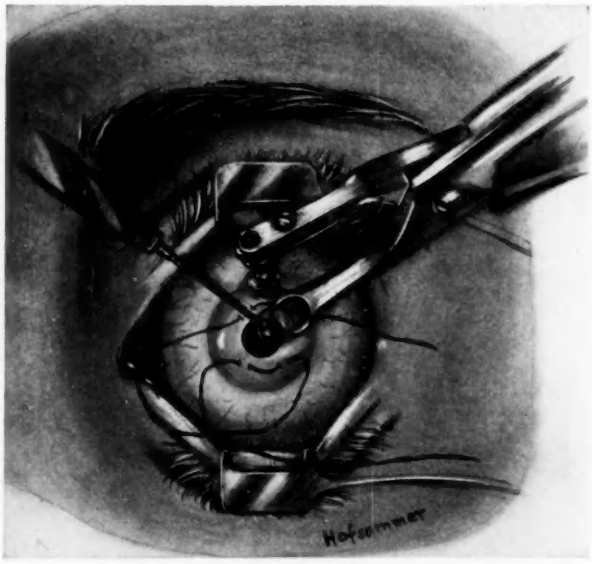


FIG. 4 — (Wiener and Alvis)

The lids are closed and sealed with a thin layer of cotton, first wet with sterile water, gently approximated to the skin of the lid and then dried with the warm air of a hair drier.

Where the cornea of the host is vascular, an erythema dose of X-ray is given the following morning, and repeated a second or even a third time if the vascularity seems to want to encroach on the graft.

Numerous animals were operated before the technic, as now used, was perfected. The work was done in the laboratory of the Oscar Johnson Institute connected with Washington University School of Medicine. Through the courtesy of Captain Minter and Commander Brown of the Navy Hospital at Balboa Park, San Diego, the technic was further advanced.

Anesthesia in the rabbit is secured by means of intravenous injection of sodium barbital with a small amount of ether added.

The suprarenin helps in the rabbits in other ways. Their aqueous is quite different from ours in that it rapidly coagulates, forming a thick, gummy mass over the wound, interfering seriously with facility of technic. The secretion of aqueous is stopped by the suprarenin which also checks bleeding in case the iris is cut.

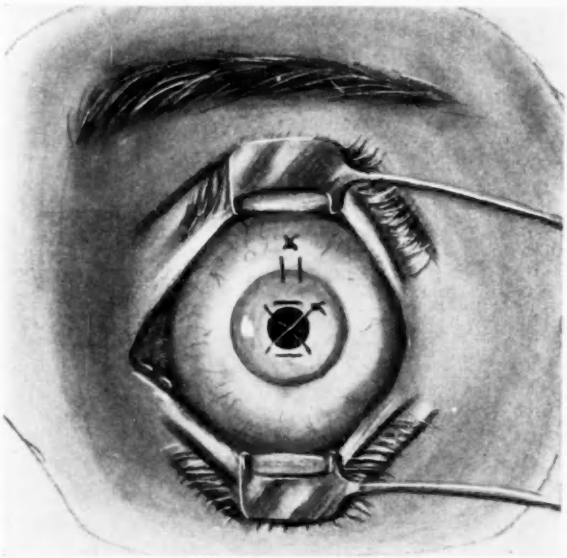


FIG. 5 — (Wiener and Alvis)

The following case is reported and photograph shown, though realizing that enough time has not yet elapsed to predict the eventual final outcome.

Mrs. L. K., age 33, housewife, first noticed poor vision at the age of thirteen. Came under the service of Doctor A. Ray Irvine at the Los Angeles County Hospital. Treated for syphilis for the last five years; Wasserman and Kahn remaining positive during all this time. Referred to the Eye Department from the Luetic service as a matter of routine. An old, inactive interstitial keratitis was revealed in both eyes. There was a general uniform corneal haze, confined chiefly to the anterior layers of the cornea. Old collapsed vessels could be seen running through the stroma. A few vessels in the deep layers, through the center. Anterior chamber was slightly shallow, some definite adhesions of the iris to the lens being made out. Good red reflex. Light perception and projection, good.

The left eye was similar to the right, except that the scarring was less extensive. Old, collapsed superficial and deep vessels could be made out. Some pigment spots on the lens. The fundus, seen hazily through the opaque media, revealed an apparently normal disc with an old area of choreo-retinitis between the macula and the disc. Vision was 20/50. Corneal transplant was recommended, although the presence of deep vascularity made the prognosis none too favorable for the right eye. Operation was performed on August 11, 1939, by Doctor A. Ray Irvine. The donor was a middle aged woman with sarcoma of the choroid, the anterior segment of her eye being found perfectly normal.

The graft seemed to have taken perfectly, the sutures being removed on the sixth day. Doctor Irvine reported that "the graft looked perfectly clear for several days after the operation, after which it became slightly hazy. When the patient left the hospital the graft was clearing. There were a few vessels in the cornea surrounding the graft, but none in the graft."

Through some slip-up in the orders, the erythema dose of X-ray, ordered the day following the operation, was not given until August 31, by which time some vessels had grown into the transplant. These had been first noticed about ten days before the X-ray was used. A second erythema X-ray dose was given on September 20. The last examination of the patient was made on this date when the following note was made by Doctor Rodman Irvine. "Vision 5/200. Slight photophobia. Eye is whitening, but still a slight amount of limbal injection persists. There is a slight, uniform haze of the transplant, with some deep striae and also some deep vessels can be seen growing into the periphery. On one side, a small strip of iris is seen incarcerated in the wound between the transplant and the cornea. At the site of the keratome incision of the cornea, there is a slight incarceration of the iris. The anterior chamber is quite shallow, being only about 1 mm. in depth. A good red reflex can be seen through the transplant, but no details of the fundus made out. The patient has had no discomfort and is quite happy with the result."

The photograph which we are showing was taken through the courtesy of Doctor A. Ray Irvine on September 11, 1939. The photograph of the first rabbit was taken September 12, 1939. The transplant was done on August 7 at the Navy Hospital in Balboa Park.

FIG. 6 — (Wiener and Alvis)

The rabbits now being shown were operated at the Oscar Johnson Institute, August 31, 1939. Photographs taken October 3, 1939. These grafts so far show no vascularity.

The advantages are obvious. The segments are rapidly obtained; they are identical, and therefore fit exactly; the bevelled edge prevents the graft from slipping into the chamber; the lens is protected so that it cannot be injured in the procedure; the stitching has been modified so that as soon as the segment has been removed from the host, the corneal wound made for the introduction of the punch is closed; a simplification of the Thomas stitch is offered. The segment is not injured, either macroscopically or microscopically by the action of the punch, nor in transferring it from donor to recipient eye.

DISCUSSION

VIRGIL G. CASTEN, M.D., Boston: About one hundred and sixteen years (116) ago corneal transplantation was first attempted. In the early years lack of both asepsis and anesthesia caused many failures. During the past century anesthesia and the technique of corneal transplantation have both greatly improved. Almost all the cases of total corneal transplantation first done by Wagenmann in 1888, where the entire cornea and some conjunctiva was transplanted, have failed.

Von Hippel, in 1888, tried so-called circumscribed lamellar corneal transplantation, but connective tissue formed at the base of the transplant, preventing visual success. He then went back to his earlier method of 1877 of partially penetrating corneal transplantation, which Elschnig later found to be the most dependable method. Von Hippel used a trephine to cut his transplants.

Elschnig modified von Hippel's technique slightly and reported many visual improvements. Filatov further modified von Hippel's operation, making a puncture and counter-puncture of the cornea, through which a strip of celluloid was passed to prevent injury to the iris and lens when the cornea was trephined.

Thomas in 1930 used a 4 to 4½ millimeter trephine, held at an angle of 30 to 45 degrees, so as to penetrate at one side. The trephine was then withdrawn and the button completed with scissors. In this way the inner surface was made smaller than the outer, giving a bevel which prevented the transplant from falling into the anterior chamber.

Castroviejo, in 1932, used a double-bladed knife, making a rectangular transplant, beveled on one side, with a keratome, and on the other three sides with scissors as he found rectangular transplants easier to bevel than round ones.

All of these operations have been time consuming. Doctor Wiener has ingeniously devised a new technique for securing uniform, mechanically obtained, beveled grafts, which can be cut in a second and transferred from donor to host. The average ophthalmic surgeon can cut uniform beveled grafts with this instrument. However, in this technique another 8 millimeter corneal incision is necessary, which has not been used in any of the former operations. This should not be a great stumbling block.

In the way of criticism, it would seem to me the instrument could be used more easily if it had a bayonette end, so that the brow would not interfere, and also an infinitely thin sleeve to prevent the graft from falling into the anterior chamber when it is cut. Further, it would seem that the graft would be subjected to great trauma and consequently there would be a higher percentage of opaque transplants by this method. This, however, remains to be seen as only one operation in a human eye is reported.

The type of suture is excellent. Conjunctival flaps have more or less been given up because of the higher incidence of infections and a suture of some type is being generally used.

RAMON CASTROVIEJO, M.D., New York City: Doctor Wiener deserves to be congratulated for his ingenious instrument, which as he claims, when put in the hands of the average ophthalmologist will permit him to obtain mechanically a bevelled graft.

In the operation described by Doctor Wiener, an incision is made at the limbus, through which one blade of the instrument is introduced into the anterior chamber. It is reasonable to suppose that this extra incision at the limbus will lead to complications such as incarceration of the iris. This supposition is corroborated by the report of the only case operated following Doctor Wiener's technique, in which incarceration of the iris took place. Of course, one case is not sufficient from which to draw conclusions. Many more cases will have to be operated upon, possibly several hundred, before definite conclusions can be drawn.

The condition of the anterior chamber must also be considered in performing a keratoplasty following Doctor Wiener's technique. If the eye is aphakic, the use of Doctor Wiener's punch may lead to luxation of the dissected leucoma into the vitreous cavity.

The simplified suture used by Doctor Wiener is just one half of the cross stitches advocated by Tudor Thomas. I have observed, however, in the illustrations showing the different steps of the technique, that the suture is placed too far from the edge of the incision. It has been our experience at the Institute of Ophthalmology, Columbia Presbyterian Medical Center, where about 200 keratoplasties have been performed, that the sutures must be placed very near the edge of the incision, otherwise, in cases where the cornea is very thin, especially in keratoconus, th cornea collapses and it is difficult or impossible to maintain the transplant in position.

When the cornea is very favorable for keratoplasty, a simplified stitch as the one advocated by Doctor Wiener may be sufficient. It is, however, good judgment to consider every case a difficult one, in order to avoid unpleasant surprises during the operation and complications during the postoperative course.

A bevelled transplant seems to be obtained consistently and easily by Doctor Wiener's procedure, and according to his observations, the edges are cleanly cut, an observation confirmed by microscopic examination.

The ingenious operation presented by Doctor Wiener deserves to be thoroughly studied. A comparative study of his and other methods of operation made on eyes of animals, will give new data to report in the future about the real value of the operation.

B. Y. Alvis, M.D., in closing: We realize that Doctor Castroviejo's last point is very well taken, indeed, and that one human case is not sufficient to draw any conclusion and that much more study is necessary, of course, both to perfect the technique and to prove its value. This paper, however, was given rather as a preliminary report, because we felt that this contribution was sufficiently valuable in that it simplified the technique of corneal transplants so much, that it would be worthwhile to give it out and let others use it and attempt to perfect the technique along with us.

The very simplicity of this technique, it seems to me, will make it possible for the operation of keratoplasty to be carried out, far away from Doctor Castroviejo and others who have had more extensive experience in this type of surgery. The length of time required for the operation is only fifteen or twenty minutes.

The statement was made that the graft would be difficult to recover from the anterior chamber. That has not proved to be the case. There were a few instances in experimental cases that the graft did slip into the anterior chamber, but there was very little difficulty in recovering it because of the large incision in the cornea. In fact, the graft is not often lost, and I do not believe it is more likely to drop into the anterior chamber than if one used a trephine or if one used the double corneal knife of Castroviejo.

The objection occurred to us that probably the graft would be crushed, but when one considers the cutting operation of this punch, one notes that the margin is cut in exactly the same way as by the scissors. A microscopic study of such a section showed no visible evidence of crushing of the corneal tissue.

THE RELATIONSHIP OF TRYPARSAMIDE REACTION TO VITAMIN DEFICIENCY

WILLIAM M. MUNCY, M.D.
PROVIDENCE, R. I.

IN JANUARY, 1934, at the request of Doctor Hugh E. Kiene, head of the Psychiatric Department of the Charles V. Chapin Hospital, I was relieved of my routine duties as Ophthalmologist to take charge of all cases of neuro-syphilis. This action seemed necessary as Doctor Kiene considered the most satisfactory treatment of this condition to be fever therapy, followed by tryparsamide. Since the fear of ophthalmic complications was an objection to the use of this drug, it seemed most important to make a thorough study of the eyes of each case before and during treatment. Our object was, first, to prove or disprove the existing opinion that any pathological condition of the eye in the present or past history was a predisposing cause of such reaction; secondly, to find if there was any condition of the patient, general or local, that indicated an incidence to a possibility of tryparsamide shock.

Since 1934 out of 152 cases of neuro-syphilis 102 had tryparsamide treatment at the hospital or at the offices of private physicians. Of these, 78 were men and 24 women, a ratio of about three to one. To date we have not known of a woman having a tryparsamide reaction.

The findings in the first forty cases treated were published in the Rhode Island Medical Journal in November, 1937. We had reaction to tryparsamide in six of the cases, or fifteen per cent. Prior to the adverse reaction five of these had normal form fields, normal visual acuity and nothing on an ophthalmoscopic examination that would indicate any disease of the tissues viewed. However, with one exception, all the forty cases of neuro-syphilis had contracted color fields of varying amounts, some being down to between the ten and five degree circle. As the one case that lacked this contraction had no signs of neuro-syphilis except a positive spinal fluid, I wondered at the time if we had not a case at the

earliest stage of cerebral involvement even before color fields were affected. At that time I raised the question whether the development of a contracted color field appearing in a positive blood Wassermann case with negative spinal test was not a presumptive sign that brain syphilis was imminent. However, in the second series, of fifty cases, to be covered by this paper, six cases of neuro-syphilis which were in the early stages had normal color fields so, whatever the value of this assumption, there are more exceptions than was previously thought to be the case. Some color fields improved under fever therapy and tryparsamide treatment, several even to normal, while others were not affected. Tryparsamide shock has no effect upon color fields unless the form field is contracted down into the color area, whereupon both fields are affected simultaneously.

Of the thirty-four cases originally reported as having no reaction to tryparsamide, some had iridic adhesions, retinochoroiditis and even optic neuritis. From these findings we concluded that the previous state of the eye was no indication of susceptibility to adverse reaction to the drug. Two of the five cases having severe reaction were returned to tryparsamide after having been built up in health and having gained in weight. They have had a number of series of treatments without further reaction to date. Therefore I was forced to conclude that there were no indications in the ophthalmic history by which one might expect an ophthalmic reaction to tryparsamide. If one does get a reaction in the presence of an already contracted form field grave results are to be expected. However, the presence of such a contracted field is no evidence per se that adverse reaction is to follow this treatment.

On reviewing all these cases I was impressed with the significant fact that at the time of tryparsamide shock there were no objective signs on examination of the eye grounds or media by the ophthalmoscope. The only sign we could elicit was the sudden contraction of the form field, which continued rapidly and then slowly improved, but fields rarely were restored to the original size. The visual acuity was seldom affected and then only slightly unless the form field was contracted down to the macula. In fact, the one case that became blind had 20/15 vision until the form fields were contracted down to less than 5°. On the other hand, the patients' subjective symptoms were most spectacular. They complained of lights and spots before the eyes and were most apprehensive of losing their sight since they bumped into objects due to

the sudden narrowing of their fields. Most patients underwent mental changes of an exciting nature, extending in some cases even to hallucinations, delusions of persecution and acute insanity, showing that the eye manifestations are only a part of a general reaction. My study of these cases brought home to me the similarity of the eye symptoms to those of toxic amblyopia, the only difference being that in one there is a central scotoma and in the other contracting form fields. One would then naturally turn to vitamin deficiency as being the underlying cause of tryparsamide shock and to vitamin "B," or "B" complex as the most probable deficiency.

From the summer of 1937 to date we have treated fifty cases with vitamin "B," and "B" complex, which was given constantly from admittance to the hospital, during fever therapy, throughout the series of tryparsamide treatments and again whenever the drug has been resumed. We have had but one reaction out of these fifty cases. This was a case of optic neuritis which, after tryparsamide shock, was brought back to its previous condition and, following numerous vitamin "B" intravenous injections has undergone seventeen treatments of tryparsamide without any further reaction. In addition, during this period, a number of cases which had already had tryparsamide reaction were sent to the hospital for treatment. These cases had not had vitamins administered as a preventive treatment but under active vitamin therapy they have had their vision restored to its previous state.

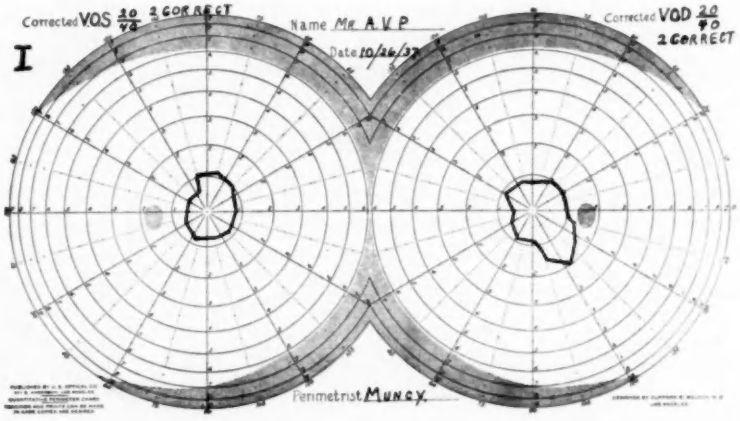
Three of these secondary cases, along with the above mentioned case of optic atrophy will now be reported in brief.

Case I. Mr. A. V. P., male, age sixty-two, admitted to the Charles V. Chapin Hospital on August 23, 1937, diagnosis of general paresis with hypertensive vascular disease. From the past history we glean the following: probably contracted syphilis between 1907 and 1910 but refused treatment when this diagnosis was later made by a physician. Ten years ago developed high blood pressure and aricular fibrillation. There is no history of alcoholism but used tobacco excessively up to 1921, when he gave it up. Present history: In April, 1937, he complained of loss of eyesight and difficulty in finding his way around, which led him to have his eyes examined. From Doctor Herman P. Grossman's records of July 12, 1937, we have the following: "Vision blurred during the last few years; anterior chamber slightly shallow; cornea clear; no deposits on posterior surface; pupils slightly irregular, do not

react to light but react definitely to convergence (Argyll-Robertson); fundus appears normal though pupils small; discs good color; vision of right eye with a plus 7.50 S. plus 1 cyl., axis 45, gives 20/40 vision; left eye, plus 8.25 S. plus 1 cyl., axis 115, gives 20/40 vision. Had impression of cerebro-syphilis and requested that the patient return for an examination of the fundus under mydriasis; ordered Wassermann test." The report on the blood Wassermann received by the patient's family physician on July 14 was found to be plus four and 400 grams of mapharson were administered. When the report on the spinal fluid of July 21 was found to be Wassermann positive, acetone plus four and cholesterol plus four, he resorted to tryparsamide. One gram was given July 23, two grams, July 30 and three grams on August 6 and 13, making nine grams in all. After the last treatment the patient complained that his eyesight was worse and said he did not know where he was even in his own home. He went to the seashore with his family where he seemed much better and was given another treatment of three grams of tryparsamide on August 20. On the 22nd he commenced to have hallucinations, both auditory and visual; thought he was going to be killed by various members of his family and became actively delirious. He also developed a tabetic gait at this time.

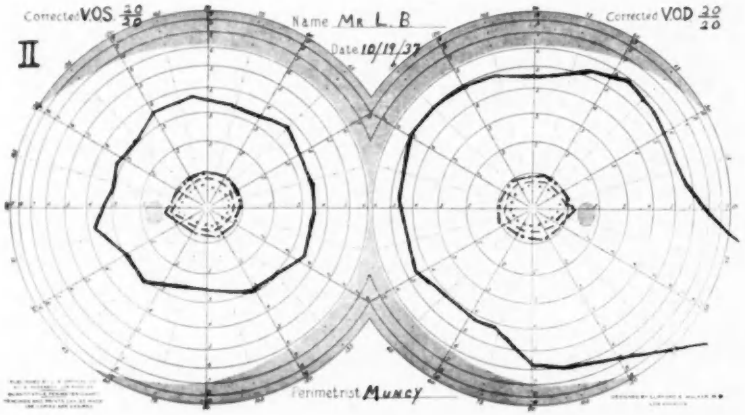
Since his condition was so alarming he was admitted to the hospital on August 23, 1937. Doctor F. Charles Hanson, our visiting oculist on service, made the following report on the 30th: "Pupils unequal, right being larger than the left, do not react to light; as far as can make out is blind in both eyes as there is no visual or mental reaction to bright light entering either eye. On dilating pupils some faint lenticular opacities were noted but outside of that the media was clear. With exception of advanced arteriosclerotic condition of the blood vessels no gross pathology can be made out." When I examined the eyes on the following day the above was corroborated and a further notation was made that where the arteries, very arteriosclerotic, crossed the veins their calibre was obliterated and the distal portions were much enlarged. There were no hemorrhages or exudates, however. He was given two yeast tablets and one teaspoonful of cod liver oil, t.i.d., from the time he entered the hospital. On September 9 he was given fifty million typhoid antibodies at 9:20 and seventy-five million at 11:30. Though this gave but a fair rise of temperature the blood pressure, which had previously been reduced, shot up

to 220 and marked cardiac involvement imperiled the patient's life so that no further use of fever therapy was attempted. By September 10 he had fair light projection and on the 21st, with his glasses on and using both eyes, he could see 2/40 but the smallness of the visual area made the taking of form fields impractical. On September 28 each eye had 20/200 and on the 26th of October the vision was 20/50 and 20/40, two letters correct, with each eye, which compared favorably with Doctor Grossman's examination of 20-40 prior to the tryparsamide shock. His form fields could now be easily taken and were found to be contracted to about 10° on



all sides. The color fields for blue and red were of the same size as that for form and green color sense was wanting. See Cut I. He was discharged from the hospital on November 5, 1937. Though we do not know what his previous form fields were, from his history and in comparison with other cases we assume that they were considerably larger prior to the tryparsamide reaction. This reaction temporarily wiped out all vision. But subsequently the previous visual acuity was practically restored, although form fields remained smaller than before the reaction. He was given sulpharsphemaine, three-gram doses, intramuscularly but tryparsamide was never again attempted. His condition remained stationary for a time but he was later admitted to the State Hospital at Howard where he died in May, 1939. On entering the hospital he had only light perception. In view of our present knowledge it is a question whether vitamin "B" intravenously, followed by tryparsamide, might not have stopped the progress of the neuro-syphilis.

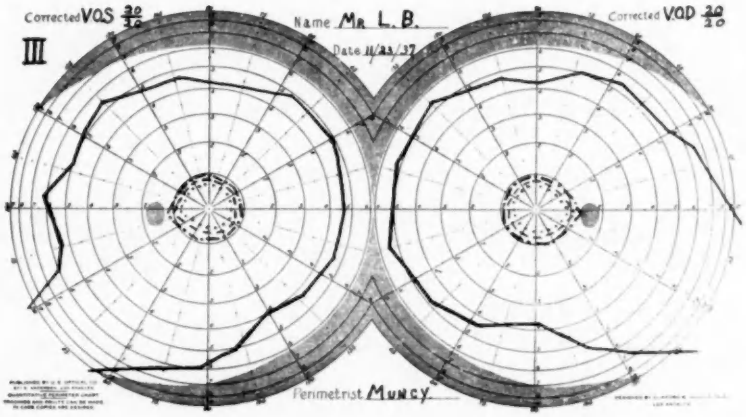
CASE II. Mr. L. B., age twenty-six, was admitted to the Charles V. Chapin Hospital on October 14, 1937. Diagnosis, general paresis. He contracted syphilis at the age of eighteen and has been under treatment at times ever since. For the last few months prior to his admission he was sullen and depressed. As he had a plus four Wassermann spinal fluid and was becoming worse under previous treatment, his physician administered tryparsamide, of which three doses of three grams were given. After the last injection he complained of not seeing well and a companion with whom he was driving said he nearly hit every car he passed and had to be con-



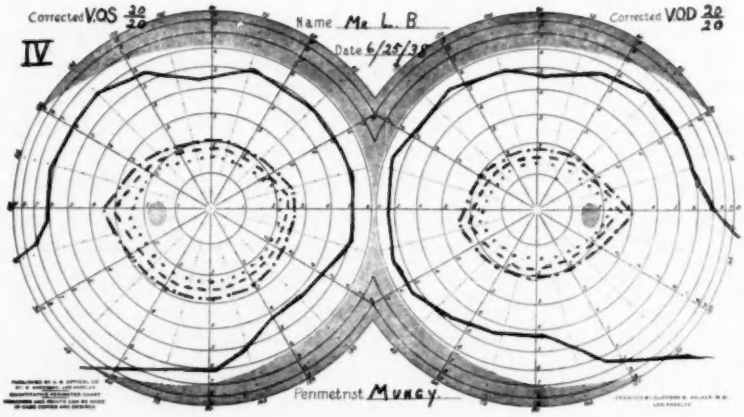
stantly cautioned to prevent an accident. The next day his vision became so bad that he could hardly see. After having two convulsions he became irrational and so violent that he was hastily brought to the hospital. On October 18 Doctor F. C. Hanson examined his eyes and reported: "Pupils equal and react to light and accommodation; media clear; discs well outlined, normal; vessels normal; no hemorrhages or exudates." I saw him on the 19th, confirmed the above and found his visual acuity to be 20/20 in both eyes. His form fields were contracted, especially in the left eye, the constriction in the right being confined to the upper temporal quadrant. He was too uncooperative to take color fields. They were taken on the 26th, however, and found to be confined to 10° and less. See Cut II.

He was given two yeast tablets three times a day from time of admittance. His general treatment consisted of eight elevations of temperature above 104° with malaria, followed by seven elevations

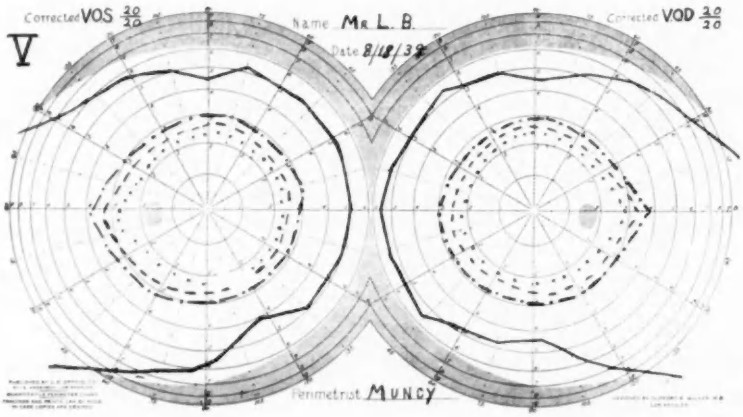
of over 104° with typhoid antibodies, as a result of which all his active symptoms subsided. A month later, on November 23, there was a considerable gain in the form field of the left eye and the right was almost normal. See Cut III.



As an examination in March, 1938, showed form fields practically normal and visual acuity normal, we advised his physician to resume tryparsamide while still continuing the yeast tablets. Form fields taken June, 1938, show but slight loss in the upper temporal quadrant of both eyes and color fields enlarged. See Cut IV. With a few exceptions, he has had three grams of tryparsamide weekly since leaving the hospital on November 23, 1937. After he left the hospital he went back to work and now has



charge of a large auto repairing shop. An examination on August 18 of this year shows even the loss in the upper temporal quadrant to have disappeared, color fields normal and vision to be one-half of the 20/15 line in each eye. See Cut V. On September 11, 1939, his spinal fluid Wassermann was plus-minus, Pandy plus-minus, Protine sixty and gold curve .0111110000, which is about normal. On September 22 his blood Wassermann was doubtful, acetone plus-minus, cholesterol plus one and Hinton positive.



On the ocular side it is interesting to note that, though the visual complaints were grave at the time of the attack, very slight actual changes were found on examination. This may have been due to the few days' delay because of the patient's want of co-operation. Although form fields do not usually return to their previous state after tryparsamide shock, in this particular instance, where the patient was under constant treatment for a period of years, we find that both form and color fields returned to normal.

Case III. Mr. G. H., age fifty-six, admitted to the hospital June 14, 1939, under diagnosis of tabes dorsalis with tryparsamide reaction. Past history shows that he contracted syphilis twenty-one years ago, at which time he was treated for two years and told he was cured. Eight years ago his blood was found to be positive and he was treated until it became negative. He used to use alcohol to excess but rarely drank of late years and was a moderate smoker. He had had lead poisoning when young and again in 1924; was a painter by trade.

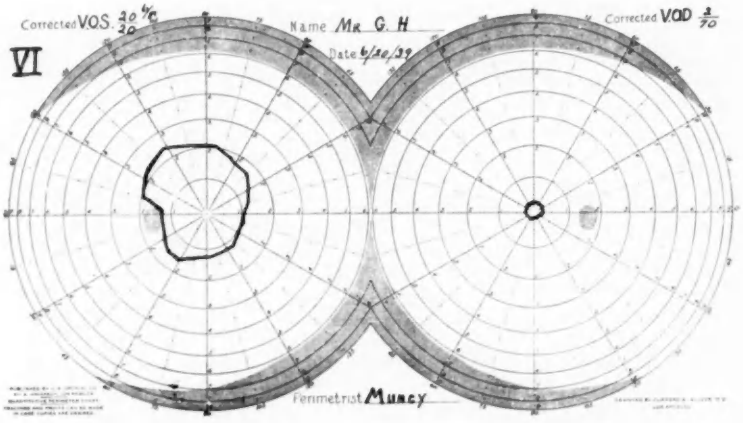
On January 7, 1939, his blood was again positive and he was given diarsenol treatments. On April 27 he went to the Rhode Island Hospital clinic, complaining of weakness of both legs with numbness. On May 16 the spinal fluid was found to be positive and on the 23rd he was given one gram of tryparsamide. He was treated again on June 6. On the 13th he returned for a third treatment but as he complained of impairment of vision it was not given and he was transferred to the Charles V. Chapin Hospital for treatment.

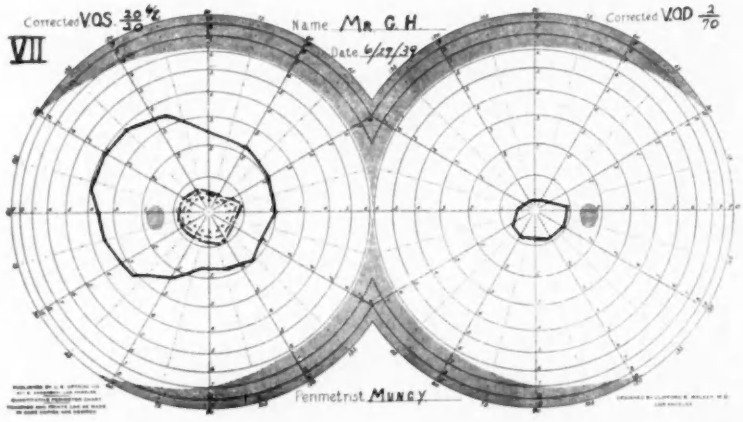
The ophthalmic history discloses that he had had an ulcer of the right eye ten years ago and was treated with satisfactory results. About three years ago he suddenly lost the vision in this eye. For a time he had to close it to see well out of the left eye but in two months he could see well with both eyes open though the vision was never again clear in the right. The left eye had always been good until the tryparsamide treatments, after which he had difficulty in seeing and going about and complained of running into things at his side.

He was given two yeast tablets and ten units of A.B.D. vitamins with each meal as well as twenty-five milligrams of vitamin "B" intravenously daily. As he was in such a weakened state when he entered the hospital typhoid antibodies were not given.

On June 19 Doctor Hanson reported: "tension normal; right cornea shows a few scattered opacities; pupils react to light and accommodation; discs well outlined and appear grayish-white. The arteries of the right eye are very narrow and terminate soon after leaving the disc, showing a general sclerosis with diffuse choroidoretinitis involving the macula region. The left, outside of a few bubbles in the lens, has normal media; disc well outlined and there is moderate physiological cupping with markings of the lamina cribrosa prominent. Arteries all tend to be narrow, as are the veins. General fundus background appears pale but no localized hemorrhages or exudates are seen."

When I first saw the patient on June 17 he could only see light and the hand of the examiner with his right eye and could not count his fingers. On the 20th he could see 2/70 and plot a small field about 3° in diameter. The left field was contracted down to 5° on June 17 but by the 20th it was 10° on the nasal and between 20° and 30° on the temporal side. The visual acuity was then 20/30 without a glass correction and six of the eight letters in the 20/20 line with the addition of a plus .75 S. See Cut VI. The next week


Corrected V.O.S. 20/20
Name MR G. H
Corrected V.O.D. 2/70
VI
Date 6/20/39
Perimetrist Muncy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Name MR G. H
Corrected V.O.D. 2/70
VII
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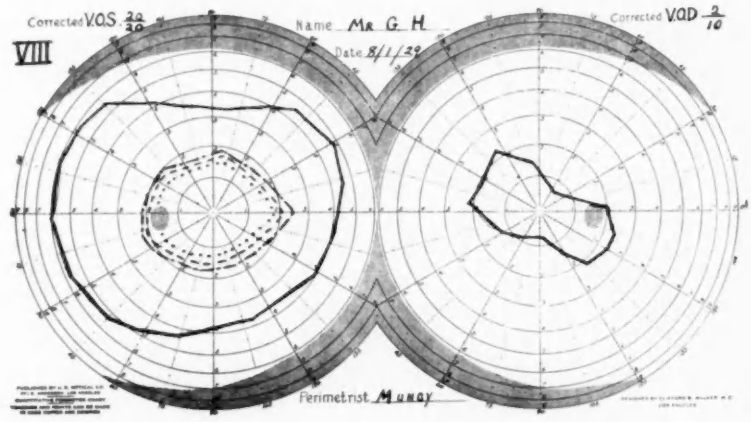
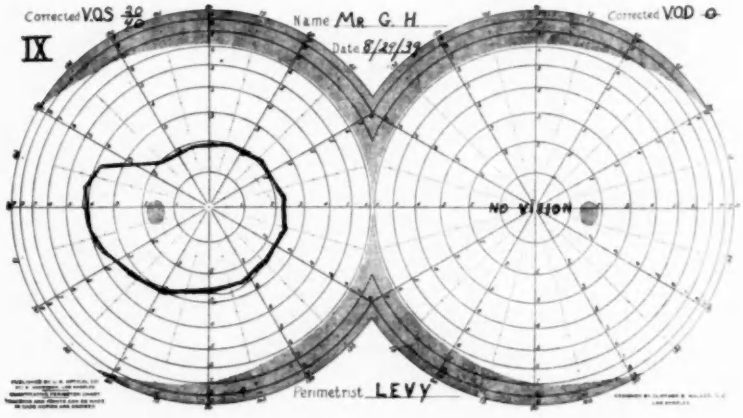
Perimetrist Muncy


Corrected V.O.S. 20/20
Name MR G. H
Corrected V.O.D. 2/10
VIII
Date 8/1/39
Perimetrist Muncy

the right eye had a visual acuity of 2/40 and a form field of 5° could be plotted. Color sense was always found wanting in this eye. The left now had a form field enlarged beyond 10° on the nasal and up to 40° on the temporal side. The color fields were found to be less than 10°. See Cut VII. By July 11 the vision in the right eye was 2/30 and both form fields had about doubled in size and on August 1st vision in the right eye was 2/10, with form field enlarged; in the left form field and color fields were much enlarged. See Cut VIII.

The patient's general health had now improved to an extent where it was felt safe to resort to tryparsamide. He was given one gram on the 7th, two grams on the 15th and three grams on the 22nd of August. During this time his eye conditions remained the same. On the 23rd he became irritable and anxious about his eye-

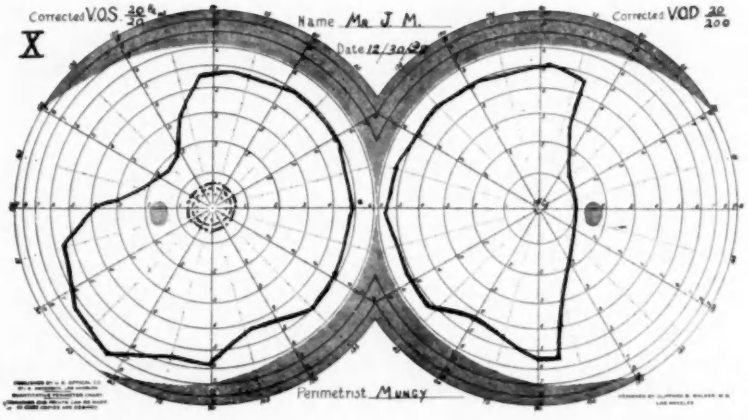


sight which he said was failing. The next day he remained in bed and refused to eat, saying he could not swallow though he took fluids without any difficulty. By the 28th he became so nervous and depressed that he needed constant supervision. As I was on my vacation at this time, Doctor Kiene ordered 100 milligrams of Betaxin, B.i.d. On the 29th Miss Levy found that the patient had no vision in the right eye. The left eye had a visual acuity of 20/40 without glasses, as compared with 20/30 prior to this attack. The form field had contracted down to about what it was on June 27. See Cut IX. That night he got out of bed and fell against the door casing, receiving a laceration of the scalp. On the 3rd of September he had general convulsions and his blood pressure ran up to

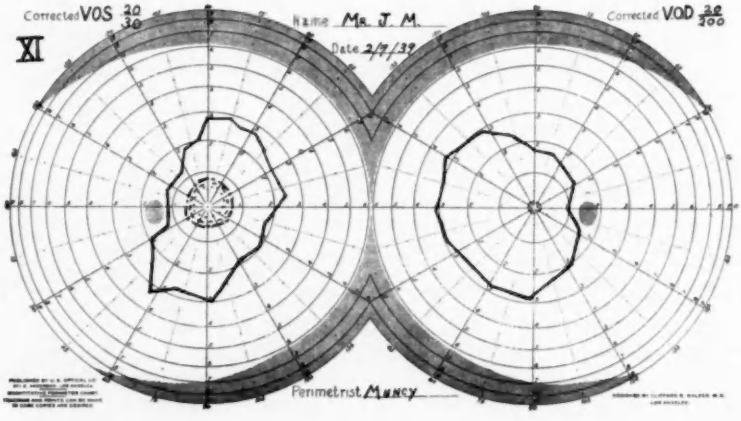
240 over 120. On the 4th his respiration became shallow and his temperature ran from 99° up to 104°. He became comatose and died on the 5th.

Doctor James T. Fallon, who performed the autopsy, found marked arterio-sclerosis throughout the brain, the blood vessels being very large and tortuous, with numerous brownish soft plaques. There were also scattered areas of anemia and soft degeneration. There were no hemorrhages. It is apparent that the patient suffered a tryparsamide shock although the extensive pathology found indicates that the disease had already reduced his life expectancy to a narrow margin at best. In this case the extensive use of vitamin "B" failed to prevent an adverse reaction due, I believe, either to the fact that the arterial disease was so far advanced or that vitamin pre-treatment was not used long enough before the resumption of tryparsamide, or to both. In the future such extreme cases will be given a more prolonged pre-treatment of vitamins before being placed on tryparsamide.

Case IV. Mr. J. M. G., age sixty. I first saw him on July 5, 1935, when he came to the outpatient department of the Homeopathic Hospital to have an eye examination for glasses. His distant vision was then 20/40 in the right eye, the addition of a plus 1.25 S. giving 20/20. The left eye was 20/50 and, with the addition of a plus 1.50 S., 20/20. I did not see him again until three and one-half years later when I found his vision in the right eye to be 20/200, which no glass would improve, and in the left, 20/70, with a plus 2 S. giving 20/30 and one-half of the 20/20 line. An examination of the fundus showed an atrophy of the right optic nerve and abnormal pallor of the temporal side of the left. Spinal fluid as well as blood gave a plus four Wassermann; acetone plus four; cholesterol plus four; and Hinton positive. On December 27, 1938, he was admitted to the Charles V. Chapin Hospital for treatment. Doctor Hanson reported, "slight ptosis of the right lid; tension normal; pupils small, do not react to light but to accommodation; media clear; discs of a grayish pallor, otherwise general fundus background appears within normal limits." My examination of visual acuity was the same as given above. There was a marked contraction of the form field in the right eye and some contraction on the temporal side of the left. In the right eye the color green was lost, red was questionable, but blue positive at the fovea. In the left there was a marked contraction of the color fields to less than 10°. See Cut X.

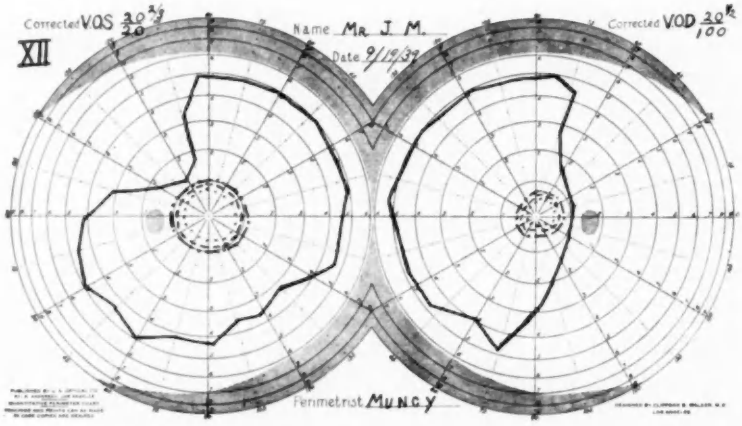


On admission he was given two yeast tablets three times a day. Starting on December 30 he was given typhoid antibodies of A. and B. organisms which gave a temperature rise of about 104°. These were continued at stated intervals until February 13, giving a total of nineteen elevations. One gram of tryparsamide was given on January 13, two grams on the 17th and 24th and three grams on the 31st, making eight grams in all. His eyes were rechecked each week and showed no change. When seen on February 7 the patient complained of poor vision and spots before the eyes and was mentally much concerned about his condition. Though the central acuity was but little affected, being one-half of the 20/30 line in the good eye, there was considerable contraction of the form fields. See Cut XI. There was an ocular nystagmus present which was very apparent on taking the fields. Tryparsamide was



discontinued. Three yeast tablets and A.B.D. capsules were given with each meal. Cevitamic acid, one gram, was given intravenously each day. In one week the visual acuity was almost back to where it was originally, the form fields were much improved and the nystagmus less prominent. By the next week the form fields had further improved, visual acuity was the same as the week previous and the nystagmus had disappeared. Pupils dilated with euphthalmine showed the fundus to be the same as previous to the attack. We discontinued cevitamic acid but continued yeast and A.B.D. capsules. Bismuth was administered for one month.

On April 11, 1939, the visual acuity was the same as prior to the tryparsamide shock. The form fields had enlarged as far as could be expected from the low of the reaction. The patient then was given twenty-five milligrams of Betaxin intravenously three times a week for three weeks. Then one gram of tryparsamide was



administered. On the fourth week two grams were injected and on the fifth, three grams. This was continued until six doses of three grams each had been given. Then the injections of Betaxin were reduced to one a week, given on the same day as the tryparsamide. Since then the patient has had seventeen doses of tryparsamide and the fields are as shown in Cut XII. Here one also notes the restoration of the complete color fields in the right eye and the enlargement of the color fields in the left.

It may have been that in this case the amount of vitamin "B" given by mouth was not sufficient to prevent the reaction experienced. The long period of high temperature may have counter-

acted the yeast intake. In the future any case having fundus changes will be fortified by vitamins given intravenously. Whatever the cause of the failure to prevent tryparsamide shock this was the only primary case having previously had administration of vitamin "B" which showed a reaction that could be demonstrated by field changes. Moreover, the action of vitamins in stopping the attack and rapidly restoring the eye almost to its former status cannot be gainsaid.

Most important is the fact that if the experience of others substantiates these findings we can now proceed to treat all cases of neuro-syphilis with tryparsamide. Furthermore, if this method of treatment can be made safe it may well be applied to all obstinate and longstanding cases of simple blood syphilis, thereby preventing them from passing over into a neuro-syphilitic state. This would place neuro-syphilis in the category of preventable diseases.

The encouragement offered by Doctor Hugh Kiene throughout the years spent on this study has added greatly to the pleasure of the work. Also the interest shown during the last year by our resident physician, Doctor Robert J. Streitwiezer, has facilitated the management of the cases. Miss Ruth F. Levy, a biologist who was appointed as acting secretary for this project and whose task it was to arrange appointments and to help in taking histories and visual fields, has been an invaluable assistant.

DISCUSSION

W. I. Lillie, M.D., Philadelphia: I presume that everyone is in accord that necessary precautions should be taken concerning tryparsamid therapy. About six per cent of these cases have untoward ocular reactions, and by far the larger portion present only the transitory symptoms, which disappear shortly after the use of tryparsamid is discontinued. A very small per cent develop a progressive loss of vision, and evidence of a simple optic atrophy may appear as a late or final result. Various procedures have been instituted to prevent such occurrences, and the ophthalmologist has played an important role in the early recognition of impending visual damage. This impending visual change is usually first suspected by subjective visual complaints, and if the use of tryparsamid is discontinued promptly the subjective symptoms usually disappear.

The oral and intravenous use of various vitamins have been of great value in many systemic diseases. The Vitamin B Complex has been of real value in some of the diseases that affect the central and peripheral nervous system. To my knowledge, the preliminary use of Vitamin B Complex to prevent any untoward visual reaction during tryparsamid therapy has been

suggested and first used by Doctor Muncy. The comparative statistics of his two groups of cases is very suggestive that the Vitamin B Complex played an important role in decreasing and practically eliminating the occurrence of any untoward visual reactions. The subsequent improvement of vision in his group of cases which previously had been affected during the tryparsamid therapy is not so significant, as vitamin therapy has been used frequently, and the results obtained are quite uniformly good and comparable to Doctor Muncy's group.

My experience with vitamin therapy for ocular conditions has been so uniformly favorable that I do not hesitate to use the combination of Vitamin B and C, orally or intravenously, as an adjunct to other accepted types of treatment. Inflammatory conditions of the retina, choroid and optic nerve respond equally well, and improvement in the retinitis of diabetes and scurvy is sometimes quite startling. The treatment of either the primary or the secondary type of optic atrophy with vitamins or any other therapeutic measure is usually of no avail, but the progressive visual deterioration can sometimes be slowed up or eliminated. At least the use of Vitamin B and C, orally or intravenously, can do the patient no harm, and may be of benefit. Its use is an addition to our present day therapeutic measures.

Doctor Muncy is to be congratulated for having originated the idea and using Vitamin B therapy as a possible preventative of the untoward ocular effects arising during tryparsamid therapy. I feel we should institute a similar regime as suggested by Doctor Muncy in our own institutions or practices, and later the sum total of our individual experience will answer Doctor Muncy's question: "Does the preliminary use of Vitamin B before tryparsamid therapy is instituted lessen or eliminate the occurrence of untoward ocular effects."

William M. Muncy, M.D., in closing: First I wish to thank Doctor Lillie for the kind reception he gave my paper. I agree with all he had to say but would like to emphasize the following points. The two cases in the first series, in which we resumed tryparsamide following an adverse reaction, in which we built up their health by a well-balanced diet and the patients increased in weight, were much slower regaining their lost fields than the later cases where vitamins were used intravenously. Furthermore, the failure in the last two cases was, I fear, due to an over-confidence in the method we were using.

In the case of optic atrophy, he was given but two yeast tablets three times a day and fever therapy was begun at once. Some writers hold that a high temperature dissipates vitamin B within the body. If so, we were probably losing rather than augmenting the vitamin body content at this time. Then to cap the climax, while he was still having high elevations of temperature, the tryparsamide treatments were started. It was notable that the active use of vitamins intravenously rapidly restored the patient to his previous condition, and when tryparsamide was again resumed, there was not a second reaction. If vitamin B complex is given by mouth much larger doses should be used.

In the case that died, though his general condition had markedly improved and his eye examination showed as much advancement as one could expect, however, on going over the cases since this paper was written we discovered the important fact that whereas the previous cases where tryparsamide was resumed all had gained in weight while this one had lost weight during his stay in the hospital.

Finally, while Doctor Lillie was discussing this paper, a number asked me if I gave yeast cakes. Yeast tablets are not yeast cakes, but concentrated vitamin B complex obtained from grains that are put out by numerous drug firms.

TREATMENT OF ACCOMMODATIVE CONVERGENT SQUINT

WILLIAM THORNWALL DAVIS, M.D.
WASHINGTON, D. C.

ACCOMMODATIVE convergent squint is an excessive convergence of the eyes due to increased accommodation necessitated by varying degrees of hypermetropia. Dissociation of the accommodation and convergence to the degree required by certain methods of training results in correcting the excessive convergence. This enables the patient to use the increased accommodation necessary to see clearly without using the excessive convergence.

In the following brief dissertation attention is invited to the fact that the correction of the refractive error is discussed only in relation to the squint.

The Diagnosis. — It is of importance to determine if the squint is purely accommodative. One may not expect to relieve more than the accommodative factor by dissociating the accommodation and convergence; hence, a careful study of the case is necessary in order to arrive at an exact diagnosis before instituting therapy.

Convergent squint is a symptom complex: The different factors in this complex must be clearly distinguished by the ophthalmic surgeon before he may expect intelligently to undertake proper treatment.

The more important factors in this diagnosis are as follows: Accommodative; neurogenic; paretic; anatomic; anomalous position of rest; vertical imbalances; anomalies of the extra-ocular muscles; disturbances of vision; pseudo-squint; high angle gamma, epicanthus, or both.

We will briefly consider the factors enumerated, not with the expectation of rendering them quite clear, but to impress upon our minds the all-important fact that but few cases of convergent squint have but one factor. They are usually made complicated by several factors which may tax our knowledge to distinguish;

hence, a routine method of examination should be followed in every case in order that we may not overlook important data necessary to a correct diagnosis.

There is no great difficulty in obtaining a cosmetic correction of convergent squint in a majority of cases. There is, on the other hand, great difficulty in obtaining a functional cure. A cosmetic correction is not a cure. An accommodative squinter who has throughout life to wear glasses in order to maintain parallel visual axes is not cured in our opinion. We, as ophthalmologists, have been prone to give children glasses in many cases where other means might be employed. Please remember that I am discussing squint only. We should bear in mind that the wearing of glasses is a physical, economic and social handicap to the wearer. We should carefully consider before prescribing glasses for children and adolescents and ask ourselves if there are other means available for the correction of the squint.

The Accommodative Factor. — At the preliminary examination careful measurement of the angle of squint, both accommodating and not accommodating, is made. This should be followed with atropine cycloplegia, using the drops for a minimum of three days. While the eyes are cycloplegic the angle of the squint should be measured again. Give the necessary correction and at the post-cycloplegic examination again measure the squint with and without correction, both accommodating and not accommodating. The purpose is to determine the accommodative factor. If there is a squint remaining after these measures have been carried out, it is probably not accommodative. Accommodative squint is the simplest form to treat in the light of our present knowledge.

The squint remaining after the correction of the accommodative factor is probably not accommodative.

The Neurogenic Factor. — This is largely of nervous origin and is exemplified by the hysterical form of squint. Other evidences of a neurosis or hysteria may be elicited. It is a confusing symptom when combined with an accommodative squint.

The Paretic Factor. — This should not be difficult to diagnose, since the squint will be of different degrees when the gaze is shifted in different directions, provided the paresis is of recent origin. The objective measurements, as practiced by Doctor James Watson White, in the different positions of the eyes, are of great

value, particularly with children, provided fixation is present in both eyes.

Anomalous Position of Rest. — Bielschowsky states that 80 per cent of all persons have an anomalous position of rest. That is to say, when fusion is inactive for any reason, the visual axes are not parallel. This may greatly modify any form of squint. It may increase, lessen, or mask other factors and is difficult to estimate.

Vertical Imbalances. — These are confusing and render difficult the treatment of the accommodative squint. One must determine with which of the classifications of vertical imbalances he is dealing, as outlined by Bielschowsky. He mentions four: Dissociated, paretic, pseudo-paretic and concomitant hyperphoria. The last three mentioned may be corrected by surgery if necessary. That first named, that is, dissociated, never. It will be necessary to determine in a case of accommodative squint whether the hyperphoria should be corrected at the beginning, during, or after the orthoptic treatment.

Anomalies of the Extra-Ocular Muscles. Complicating accommodative, or any form of squint, is confusing and difficult of diagnosis. Only a thorough knowledge of the subject and a searching examination will reveal them.

Disturbances of the Vision. Diseases of the percipient, the transmissive or the receptive elements of vision, resulting in disturbances of the visual acuity in early life, particularly in infancy, coupled with an anomalous position of rest, will produce a squint to which an accommodative element may be added. In the absence of fusion due to monocular vision from reduced visual acuity in one eye, orthoptics is not helpful, unless and until the amblyopia is corrected. Surgery may give a cosmetic cure.

Epicanthus, high angle gamma, and anomalies of conformation of the orbits may startlingly resemble squint. The diagnosis should not be difficult. Such anomalies in the presence of an accommodative squint complicate the treatment insofar as the attitude of the child's family is concerned.

Treatment of Accommodative Squint. — Twenty years ago we advocated the withdrawal of glasses in the treatment of squint and affirmed that a squint was not cured if glasses were necessary to keep the eyes straight.

For fourteen months we have been using the method as practiced by certain English ophthalmologists in the Central London Ophthalmic Hospital and other London hospitals. We find its efficacy quite remarkable in purely accommodative squint. It is their experience — and ours also — that approximately three diopters of hyperopia may be overcome; that is, if the child requires four diopters for his full correction, this would be given him in the beginning. Teaching him to accommodate three diopters without converging, that is to say, dissociate his convergence and accommodation to this degree, would make it theoretically necessary for him to wear plus one diopter to see clearly. As a matter of clinical experience we find he will require no glass for distance and will see satisfactorily with the eyes straight and no symptoms. He may or may not require glasses for near work, depending largely on his physical vigor and the amount of near work he has to do. Astigmatism may require correction for near work and possibly for distance.

If amblyopia is present total occlusion is necessary until the vision in the amblyopic eye is 20/40. The vision in the fixing eye is then reduced to that of the amblyopic eye by means of scotch tape on the lens or by other means. This enables the patient with accommodative squint to attain binocular vision most of the time, thus stimulating fusion and hence dissociation of the accommodation and convergence.

Total occlusion may produce an alternating squint which protracts the dissociation of the accommodation and convergence, and hence delays the cure of the squint. It may, however, be necessary to use total occlusion, since the amblyopia must be corrected. It is necessary also to overcome abnormal retinal correspondence. The squint cannot be cured until this is done.

The patient is taught that he must keep his eyes straight, though the vision be blurred. Hyperopes of three or four diopters are requested to go without glasses for distance, keeping the eyes straight and vision blurred. Shortly the distance vision begins to clear, that is, the child is learning to accommodate without using his convergence. The first lesson the child learns is to be able to distinguish between clear and blurred vision. Blurred vision means the eyes are straight, while clear vision means one eye is converging. The child practices this many, many times on the visual acuity chart at six meters until he understands and gets the feel of knowing every

time he squints. This can be taught to most four-year old children but scarcely younger.

Hyperopes of four diopters or less are requested to discontinue wearing glasses for distance two to four hours daily, when they have learned to accept blurred images with eyes straight. Later they dispense with glasses entirely for distance. Since cooperation, intelligence, and demands for near work are variable, the length of time the child goes without glasses will vary. He should go without glasses as much as he can and must form the habit of keeping the eyes straight without them. At the beginning of treatment glasses are worn for near work.

The mother is given a visual acuity chart of letters or pictures for home work with the child. She sits facing the child, watching his eyes closely. With eyes straight without glasses he can read probably 20/200. She frequently covers first one eye and then the other to be sure there is no squint. Hyperopes of three diopters learn to read 20/20 with eyes straight without glasses in a few months. Those with amblyopia or alternating squints take longer. When the patient can accomplish this we may consider the squint corrected for distance.

To control the squint for near is more difficult. Bar reading with glasses daily at home and frequent checks by the orthoptist are carried out. With good distance vision without glasses and without squint bar reading may be commenced without glasses. Frequent checking by the surgeon and the orthoptist are absolutely necessary. If the bar reading is not being done correctly, it is worse than useless.

In the office the patient is trained on the synoptophore or similar instrument, the cheiroscope, the diploscope, the Remy separator, and bar reading. His hyperopic correction is reduced by one-half to one diopter at a time until he can read easily Jaeger 1 or the equivalent without glasses and with straight eyes.

The treatment of accommodative squint is difficult, requiring most careful study for diagnosis and long, meticulously correct observation and treatment by the surgeon and orthoptist. Without intelligent understanding and enthusiastic support by all concerned, it is useless to begin the treatment. Obviously this excludes many from this method of treatment.

DISCUSSION

GEORGE P. GUIBOR, M. D., Ottawa, Ill.: I believe that we are indebted to the essayist for emphasizing in this paper the type of strabismus which is most amenable to non-surgical treatment. This type, which is practically universally called "accommodative esotropia," is the type of convergent squint in which the deviation is less for distance than for near. It is improved by the wearing of convex lenses, usually a plus 3 diopter sphere with a cylinder, by the use of atropine and by fusion training.

The essayist has emphasized, too, that squint is a symptom complex and that several etiologic factors are associated with most cases of strabismus. In a similar way he calls our attention to what he considers a cure of strabismus. There should not only be a cosmetic recovery from the deviation but also a functional recovery. Thus the visual axes should be parallel for distant vision and for near vision and the fusion power should be normal. As a matter of fact, however, few normal individuals have perfect fusion without fusion training. We should, therefore, not expect every patient with parallel visual axes to have perfect fusion ability. Most of them, however, have good binocular vision, but many times have imperfect stereoscopic ability.

Like all those interested in motor anomalies, the essayist reiterates that the most important factor in securing a satisfactory recovery from the deviation is the diagnosis of the type of squint. He also discusses the diagnostic routine and here, too, impresses upon his readers the importance of determining the degree of deviation for distance and for near with and without corrective lenses, and again repeats these measurements when the eyes are under a cycloplegia and in the different directions of gaze.

One interesting sentence in this paper especially attracts my attention. This sentence is as follows: "As glasses are a physical, economic and social handicap, one must know that other means are available for the correction of the squint or for the relief of symptoms." This is talking about accommodative esotropia. This sentence to me suggests that orthoptic fusion exercises and surgery can be used in place of glasses in treating accommodative esotropia. I have attempted this several times, but have always felt that I was not doing the correct thing by stimulating convergence indirectly through the accommodation and that this was a more difficult system of treating neuromuscular anomalies than when accommodation was kept under control by glasses and atropine. But I shall have to revise my attitude concerning this routine of removing lenses and attempting fusion exercises when such men as Doctor Davis and those of the London school of orthoptics follow this technique.

In conclusion the essayist emphasizes the following causes for failure in treating accommodative esotropia: First, a severe heterophoria or anomalous position of rest coexisting with the squint; second, a vertical imbalance; third, visual defects such as amblyopia; and, fourth, anomalous correspondence.

I have three slides which emphasize my short experience with accommodative esotropia associated with some of these causes for failure.

(Slide) This patient had a right esotropia of 50 degrees without glasses and 40 degrees with glasses. That was for distance and for near. I think that was due to a spasm of the right internal rectus, and to overcome that spasm I employed a horizontal prism, base out, in front of the right eye, and we can see the effect of that prism. So that one of the questions I should like to ask the essayist is what does he do when a patient has a spasm of convergence or a spasm of one internal rectus, or a spasm of accommodation, when he attempts to treat such patients by the method that he follows. So one of the methods that I have found in the treatment of accommodative esotropia associated with convergence spasm is the use of a prism, under-correcting, of course, the degree of spasm as determined by the occlusion or screen test.

(Slide) This slide demonstrates an anomalous position of rest as follows: In the first place, the first picture to the left discloses that this patient has 30 degrees of left esotropia without his glasses and 25 degrees with his glasses. After resecting both external recti this patient has an apparent parallelism of his visual axes, but the point about the anomalous position of rest is that under cover, though I did not touch the internal recti, this patient at the present time has an exotropia of 10 prism diopters, by the mere resection of both external recti. So that this patient had a convergent squint with a convergence insufficiency which we often fail to recognize. This convergence insufficiency which he has at present was made manifest merely by resecting both external recti.

(Slide) This demonstrates in my short experience the effect of eccentric fixation in an anomalous correspondent upon the result that we can obtain by non-surgical treatment. These two boys are brothers. One boy, the younger, at the left, is seven years of age. The older boy, at the right, in the first group of two pictures, is thirteen. We see by the corneal reflex that both patients have similar degrees of deviation. They do not vary more than 5 degrees, not more than 10 prism diopters. The other interesting factor is that both of them have similar lenses before each eye, plus 3.00 D. Sph. with a plus 1.00 D. Cyl. at 90, and yet when we note the lower two photographs we see that the older boy has only 5 degrees of convergent squint remaining. In the younger boy, however, the deviation has persisted. The visual acuity in the older boy has improved. By the way, the visual acuity in both of these boys was approximately 20/40. The visual acuity in the older boy has improved to 20/20. The younger boy's amblyopia has persisted. The only explanation I can offer is that because of the eccentric fixation of the younger boy, there is an anomalous correspondence which I have been unable to overcome.

So these three patients from my viewpoint demonstrate, first, the spasm of convergence (or the spasm of one internal rectus in this case), the second, an anomalous position of rest (a convergence insufficiency), and in this case an eccentric fixation.

GRADY E. CLAY, M.D., Atlanta, Ga.: The author has stressed the most important factor in the treatment of accommodative convergent squint, namely, orthoptics. The treatment of this type of squint has progressed

very little during the past decade. For years the refractive error alone was corrected. Later some men attempted to obtain a cosmetic result by surgery. Only within the past twenty years have most ophthalmologists insisted on surgery as soon as it has been seen that glasses would not correct the squint. There are a few bold enough to insist on surgery before any attempt is made at wearing a correction.

It is true that the parents of a squinting child are primarily interested in a cosmetic result, but as ophthalmologists we should be most interested in the development of binocular vision, and I should like to ask how one can be sure of ever obtaining fusion without the use of orthoptic training. It is true, and most unfortunate, that the men who discredit orthoptics are those who have not earnestly sought its true value.

Most cases in whom we develop fusion with good amplitude of accommodation have very blurred vision at first which clears with training but in our opinion to start the treatment of such a case with the whole idea of removing the glasses is not a good practice. In cases of accommodative squint with small errors this may be done easily. In case of plus 2 diopter sphere with small astigmatic error, which straightens up after the correction is worn, can be trained to develop fusion with good amplitude in a short period and such a child may in the course of time leave glasses off without discomfort. Many of these cases which Doctor Davis has reported are those in which a high error exists. In these cases with high astigmatic error I do not think it should be attempted. If we should adopt such a procedure, it seems to me there is no reason to give a patient a correction. However, for girls it is most important that this be attempted, and I feel quite sure that in the large majority of cases they can be trained, because of the interest in trying to develop fusion and in holding their eyes parallel without glasses. In this particular type I would certainly recommend that an effort be made with the individual, with the hope that she may, for social purposes only, leave her glasses off.

WILLIAM THORNWELL DAVIS, M.D., in closing: I thank my colleagues very much for their discussion. For a subject as intricate as this it is utterly impossible to give the many, many details.

Surgery should not be resorted to in purely accommodative squint. It is absolutely contraindicated. If operation is done there is very likely to follow an exotropia for distance and an esotropia for near. I have had this unfortunate experience occur in one of my cases. If there is residual squint following the correction of the accommodative squint the residual portion may be corrected by surgery.

In cases with anomalous retinal correspondence, our experience has been that they are very dangerous, indeed, to do surgery upon. In some cases the squint cannot be cured. If one disturbs the convergence system in such cases one is likely to get a wide swing in the opposite direction and have a greater exotropia than there was an esotropia.

In regard to what Doctor Clay said about the use of glasses, I am entirely in sympathy with that. Of course, with high errors, particularly of

astigmatism and high errors of spherical hypermetropia, the child will need glasses, but I was speaking, mind you, of correction of the refractive error in regard to the squint only. I did not enter upon the necessity of glasses for other purposes. Obviously, they may be necessary, but what I meant to say was that we could cure these accommodative squints with high refractive errors, so that it is not necessary for them to *wear glasses to correct the squint.* Perhaps I did not make that clear.

(Slide) While you are reading the slide, I will give you the statistics on accommodative convergent squint. Of 98 cases seen in our private clinic from July, 1938, to September, 1939, 21 were accommodative squints. The number on active treatment at this date is 7. Because of insufficient cooperation 6 out of the 21 cases were uncompleted. There were 8 cases out of 21 who are considered correct, that is eyes straight without glasses, which is approximately 26 per cent. It is a rather low number but you do not get very many purely accommodative squints.

This is an important point. Of those included in the number corrected, 3 had relapses two to three months after they were considered corrected. It was necessary to institute active treatment again in these cases and they very promptly responded to this treatment. So after treatment of accommodative squint the cases should be kept under observation for some time.

VISUAL TESTING METHODS IN SCHOOLS

JOHN B. HITZ, M.D.
MILWAUKEE

INTRODUCTION

THE OBVIOUS, but at present unattainable, ideal is for every school child to have a yearly ocular examination by a competent ophthalmologist. Inasmuch as this is impractical, some type of screening test must be carried out in the schools, by either lay persons or the attending general physician. The following study is an attempt to evaluate some of the present methods used in the schools by a comparison of the following:

1. The Snellen Chart.
2. The Betts test.
   (a) As interpreted in Betts' textbook on reading difficulties, published in 1936; and
   (b) As interpreted by the Department of Child Welfare Independent School District of Davenport, Iowa.
3. A group of ophthalmic tests consisting of the Snellen Chart at six meters, the Duane Screen Test at six meters and thirty-three centimeters and the Worth Four Dot Test at six meters and thirty-three centimeters. This group of tests will hereinafter be referred to as the author's group of tests for lack of a better name.

DESCRIPTION OF TESTS AND PROCEDURE

The Snellen Chart was used under uniform conditions of lighting of approximately 20 foot candles. 20/30 or better was considered as passing.

The Betts tests which, for the benefit of those not familiar with them, consist of a series of twelve stereoscopic slides used in a calibrated stereoscope and are designed to examine the following functions:

1. Gross fusion.
2. Distance fusion.
3. Visual acuity of both eyes and each eye separately.
4. Vertical muscle imbalance.

5. Depth perception.
6. Lateral muscle imbalance.
7. Fusion at the reading distance.
8. Sharpness of image. (The latter test being one based on the principle of the astigmatic dial.)

These tests were used and graded according to Betts' instructions as described in the 1936 edition of his textbook on Reading Difficulties.

These same tests were then regraded according to the Davenport, Iowa, method which is a percentage system, deducting 5-10 per cent for each failure of an individual test, 75 per cent being considered a passing grade.

In the author's group of tests the Snellen Chart was used as described above, 20/30 or better being required for passing. Fusion of the Worth Four Dot Test at six meters and thirty-three centimeters was required. In both the distance and near test, each dot subtends a visual angle of 13.3′ and the horizontal and vertical distance between dots an angle of 40′. In the use of the Duane Screen Tests for six meters any consistent movement of the eyes, in or out, on alternate covering was considered failure to pass. In the Screen test at the reading distance any outward movement of the eyes was considered failure and any movement of the eyes inward not neutralized by a five diopter prism, base in, was considered failure. Visual acuity tests at the reading distance were not included because of the usual large amount of accommodation present in children and the relative rarity of accommodation paralysis. Fusion tests were included because of the increasing evidence that fusion and ocular dominance are important factors in reading disabilities and, secondly, because they are a good check on the visual acuity and muscle balance tests.

The tests were all performed on average school children between the ages of eight and sixteen. Seven hundred and forty-five examinations were performed in three different schools by three Lay Examiners, untrained except for two to three hours' individual instruction in the use of the various tests. The tests were performed on all the individual children at the same session. The order in which they were given was alternated in order to exclude fatigue phenomena. It was understood that none of these tests were to be considered in any sense diagnostic, but were merely methods of determining which children should be referred to an ophthalmologist for a complete examination.

SLIDES — RESULTS

SLIDE 1

Reproduction of Betts Stereoscopic Cards

Number of Cases Examined: 745

	Pass	Failure
Snellen Test	89.7%	10.3%
Betts Test (Betts Grading)	17.4%	82.6%
Betts Test (Davenport Grading)	58.4%	41.6%
Author's Group of Tests	55.4%	44.6%

At once can be noted the marked discrepancy in results. It would seem obvious that any test which finds 82% of school children with defective vision is far too discriminating and defeats its own purpose. If 82% of school children have defective vision it would be far more sensible to refer all children to an ophthalmologist in the first place and eliminate a screening test.

The figures further suggest that the Snellen test used alone is missing a considerable number of muscular and fusional difficulties.

Comparison of Snellen Test and Betts Visual Acuity Test

Number of Cases Examined: 745

Tests Agreed	Failed Betts Passed Snellen	Failed Snellen Passed Betts
90.5%	8.0%	1.5%

With the visual acuity test the correlation is fairly good. It would seem that the Betts test was slightly more discriminating.

Comparison of Fusion Tests

Number of Cases Examined: 745

Tests Agreed	Failed Betts Passed Four Dot	Failed Four Dot Passed Betts
53.5%	35.3%	11.1%

The correlation here is not good and the Betts test again seems to show the greater percentage of failures.

Comparison of Muscle Balance Tests

Number of Cases Examined: 745

Tests Agreed	Failed Betts Passed Duane Test	Failed Duane Passed Betts
68.9%	26.4%	4.5%

Again the same comment can be made concerning these tests as was made concerning the fusion tests.

The reason for the lack of correlation in the fusion and muscle balance tests, in my opinion, is that the introduction of a stereoscope produces artificial factors which inhibit normal binocular vision. Anyone who has spent any time attempting to fuse stereoscopic x-ray plates will testify that it is not always easy. Further, a psychic factor enters, in that the object viewed is actually only a few centimeters from the eyes, although due to the decentered lenses of the stereoscope optically the object is at infinity.

A further criticism of the Betts test is that the time consumed in examining the individual patient is almost double that of the author's group of tests. The correlation between the author's group of tests and the Davenport method of grading the Betts test was quite close. Here, however, in addition to doing the Betts tests which in themselves are time consuming, one has to grade each test according to a relatively complicated percentage system.

SUMMARY AND CONCLUSIONS

The ideal school visual test inasmuch as it seems impractical to have all children examined by an ophthalmologist, would seem to be one which detects the majority of ocular defects which might have a bearing on health or educational problems. According to the figures it is apparent that the Snellen Chart used alone misses a certain percentage of muscular and fusional difficulties. It is further apparent, I believe, that the Betts test has two major faults in its present state of development. First, it is definitely too discriminating a test. To repeat, if over 80% of children have ocular defects the sensible procedure would be to have all children thoroughly examined without a needless preliminary screening examination. Second, the figures obtained from the Betts tests are not well correlated with those obtained from the use of more generally accepted ophthalmic tests. If visual screening tests, introducing different principles than those used by the large majority of ophthalmic physicians, are to be used in the nation's school system, the ophthalmologic profession has the right to expect that the findings will fairly well agree with the more commonly used procedures.

The author's group of tests would seem to strike a mean between the other two tests considered in this paper. The tabulated results would seem to warrant the conclusion that the tests in present use are either inadequate or over zealous and that a further effort toward the development of standard visual efficiency tests to be used in the nation's schools should be made.

DISCUSSION

A. D. RUEDEMANN, M.D., Cleveland: First I should like to congratulate and compliment Doctor Hitz on attempting to work out the problem of screening out the school children. Secondly, I should like to ask the question: for just exactly what are we striving in our examination of school children? Are we attempting to make a decision as to their future eye work? Are we attempting to make a diagnosis, for after all we are deciding or are attempting to decide, in the school examination whether that youngster is or is not entitled to further eye examination. And just exactly how important is this complete muscle examination without a good basic atropine refraction? Certainly, it is ideal to have an atropine refraction. If it is ideal to have an atropine refraction, then the atropine refraction should be attempted and all these various muscle tests should not be done until that is attempted.

The lack of correlation between the author's tests and those of Betts might lie in the fact that the author does no use a vertical muscle test or a test for stereopsis. You will note in the tests of Doctor Betts he has used any number of different cards to screen out the various muscle errors. It may be that if we used more tests we would have more error. The fact remains that the youngsters are not equal to the muscle tests in most instances until they reach the age of eight or nine years, and probably we are trying to make up for an inadequate school system or inadequate teaching by trying to prove that most children have poor eyesight rather than poor teaching.

It would seem to me that the proper procedure would be one of finding out if the youngster has a visual defect, has certain clinical signs or symptoms, such as headache, dizziness, and arrest, and that the question of reading difficulty should be left for a competent oculist. I do not believe that we can turn over to any lay people the decision as to whether this youngster should carry on with his reading or whether this youngster should be sent to an oculist or not. I think the decision made so early in an individual's life is one of too much importance to be left for a lay person with two or three hours of training on a few tests. Certainly, if the oculists are not competent to do it, a lay person is less so.

Further, I believe that as we study these individuals we will find that much of our trouble lies with congenital ocular defects. Some of these youngsters are mentally deficient and that also should be screened out. Then there are psychiatric problems which are also involved. I do not believe, personally, that the screening out of muscle errors is the answer to the problem of visual testing in the schools. I have seen a great number of these youngsters who have been tested by the Psychiatric Division and those who have been studied because of their reading handicaps, and it is surprising that all of them do have refractive errors and muscle errors, but the refractive error and the muscle error is not the cause of that youngster's dumbness or his lack of ability to get along in school.

We are missing the ball some place because we do not insist that these youngsters be properly examined and that every youngster arrive at the school door with a card in his hand saying what his refractive error is by

good retinoscopy or by good, complete examination. We should not turn over the early decision as to this youngster's eyes to a lay individual.

It seems that this may be ideal, but we have a long ways to go, because Doctor Parker told me this morning that twenty-five years ago they started the school examinations in Michigan, and in twenty-five years we have reached the stage where commercial organizations are doing most of the lay testing. I believe that we should go still further and insist on medical ophthalmologists getting into the school systems, one way or another, and working out a series of standard examinations by which a teacher can decide whether that youngster should go through regular school or be put into one of the sight-saving classes.

After all, the school systems are to blame for a great deal of the youngsters' difficulties in starting them to learn to read too early. It is not necessary that youngsters at the age of six, in the kindergarten years, be taught to read. They are not mentally equipped to do it at this time, and it would be much more advisable for the effort in all this work, which adds to the school budget, to be placed in the younger grade groups, allowing them more time for play and less time for visual effort, for which they are not visually or physicially equipped.

I think that if we are to study these youngsters from the point of view of complete muscle examination, as advocated by the Betts' test, then I believe that the youngster is also entitled to a complete refraction, which after all is the basis of all good eye examination.

JOHN B. HITZ, M.D., in closing: I should just like to make a further plea for standardization of tests, which we now use or which we approve. If we did nothing more than to standardize the lighting on the Snellen chart, I think we would have done a great deal. I think it is quite obvious that there is no good substitute for a careful eye examination or for a careful, trained observation of the children as they do their work in school, and a careful listing of symptoms, etc. Any screening test in the schools is obviously at best a filler-in until the time we can examine all of these children thoroughly and individually.

PULSATING EXOPHTHALMOS*

SAMUEL J. MEYER, M.D., *and*
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BY INVITATION
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SINCE the first clinical description of the condition known as pulsating exophthalmos by Benjamin Travers[1] in 1809, there have been 621 cases collected up to 1927. Harkness[2] felt that "a condition of such rarity and with such striking symptomology is deserving of record whenever encountered."

The authors have reviewed the literature since Harkness' report, bringing the total of pulsating exophthalmos cases to 786 (to 1939) and have added six of their own.

Of the several anatomical causes of pulsating exophthalmos, the most frequent is traumatic rupture of the internal carotid artery in its passage through the cavernous sinus. Rawlings[3] stated that 70% of fractures of the base of the skull involve the body of the sphenoid. Both the internal carotid artery and the cavernous sinus are comparatively immobile in this region so the underlying fracture might rupture or injure their walls. Penetrating wounds may also rupture or weaken the arterial walls. Next most frequent as a cause of this condition is a spontaneous arteriovenous fistula between the internal carotid artery and the cavernous sinus, most frequently due to generalized arteriosclerosis. Lues has occasionally been a predisposing factor. Other causes are vascular tumors of the orbit or surrounding structures, simple aneurysm of the ophthalmic artery, arteriovenous communication between the internal carotid artery and the internal jugular vein at the entrance of the carotid canal, arteriovenous communication in the orbit and absence of a large part of the orbital roof.

These cases exhibiting the syndrome of oculo-orbital pulsation are best divided into two groups, as follows:

(1) True pulsating exophthalmos, including (a) carotid-cavernous aneurysms, (b) intra-orbital aneurysms, either arterio-

* From the Illinois Eye and Ear Infirmary.

venous, or of the ophthalmic artery, and rarely (c) arteriovenous aneurysm between the internal jugular vein at the entrance into the carotid canal, and (d) simple aneurysm of the internal carotid artery.

(2) False or pseudo-pulsating exophthalmos, including orbital angioma, meningocele, and absence of a large part of the orbital bony roof.

In studying the etiology in 110 cases in which the etiology was stated, 83 were found to be traumatic, and 27 spontaneous.

The average age in the spontaneous group was 44.2 years and in the traumatic, 36.4 years. Only the cases of vascular origin are considered. In the traumatic cases, males predominated 2.2 to 1 (53 males to 25 females). In the spontaneous cases the proportions were practically equal (14 females to 13 males).

MECHANISM OF ORIGIN OF TRUE PULSATING EXOPHTHALMOS

Following the rupture of the internal carotid artery there is an overflow of arterial blood into the venous sinus with consequent increase in the venous pressure and reversal of venous flow in the ophthalmic veins and their tributaries. The resulting stasis, edema and increase in blood volume in the venous bed produces exophthalmos. Through the dilated venous channels the arterial pulsation is transmitted to the eyeball and orbital tissues. Since the ophthalmic veins are the only tributaries of the cavernous sinus which have no dense tissues around them, such as is present around the intracranial venous sinuses, they may dilate and give rise to the pulsating large venous masses often seen at the internal angle of the eye.

In the spontaneous cases it is believed that weakening of the vessel walls or rupture of simple aneurysms are the chief direct causes of the arteriovenous fistula.

CLINICAL PICTURE OF CAROTID-CAVERNOUS ANEURYSM CASES

The most frequent history is that following a severe trauma; there is a period of unconsciousness, on recovery from which there is a swishing noise in the head, often failure of vision, and unilateral exophthalmos may occur simultaneously, with the bruit, but more frequently follows it. Vertigo may be an annoying symptom in some cases. All symptoms usually tend to be exaggerated by stooping and any physical exertion.

Objectively, pulsating exophthalmos is characterized by proptosis of one or rarely, both eyeballs. There frequently is a loss of motility of the globe in one or more directions, most frequently laterally. Palpation of the lid-covered globe reveals a thrill. It may be necessary to apply slight pressure to the globe to elicit pulsation which may or may not be obvious. The stethoscope reveals a swishing bruit synchronous with the radial pulse, diminishing or disappearing with pressure over the common carotid in the neck on the involved side. There is often a marked swelling of the lids and chemosis of the conjunctiva, especially the lower palpebral portion, which may evert the tarsal portion of the lower lid. The bulbar conjunctiva may reveal a Medusa-head-like engorgement and tortuosity of the veins. The cornea may be affected by lagophthalmos. The arteries and veins of the retina are almost constantly engorged and tortuous. In a case reported by Krattan[4] the condition had been present for twenty-five years; yet the central vein was normal. Kattan explained this by the assumption that the central vein may pour its blood into the inferior ophthalmic vein instead of the superior, hence the blood may pass to the pterygoid plexus and not reach the cavernous sinus. Edema of the disc margins, retinal hemorrhages, clouding of the vitreous or optic atrophy may be found. Venous masses at the inner canthus may occur. The lesions tend to be progressive although some have cleared up spontaneously.

The first eight cranial nerves, especially the sixth, may be involved by the direct effects of the original trauma, by the direct pressure of the dilated vessel, hemorrhage, or by the venous congestion and stretching due to the exophthalmos. The supraorbital nerve may be involved by pressure by the pulsating superior ophthalmic vein and its tributaries at the exit of the nerve from its canal.

According to de Schweinitz 20% of all cases terminate in blindness and no more than 11.1% have retained normal vision.

GLAUCOMA IN PULSATING EXOPHTHALMOS CASES

Hudele[5] in 1928 classified pulsating exophthalmos in the group of causes of secondary glaucoma together with other cases in which venous stasis was present. This group consists of thrombosis of the central retinal vein, cyanosis retinae, cavernous sinus thrombosis and facial angioma in addition to pulsating exophthalmos. He cites the changing proportion of cases in which glaucoma was

found since the clinical entity of pulsating exophthalmos was recognized.

DIAGNOSIS

In order to obtain accurate localization of the site of the lesion, several corroborative laboratory tests have been made. Terry and Mysel used 25 cc. of thorium oxide intra-arterially in one case and obtained x-ray evidence of an arterio-venous fistula between the internal carotid artery and internal jugular vein in the neck. This method was attempted unsuccessfully twice in one of the authors' cases.

Pichini[6] used potassium iodide successfully in angioencephalography to diagnose a carotid-cavernous fistula. Brown[7] uses chemical determinations of the oxygen content of the blood of the regional veins to make the diagnosis of arteriovenous aneurysm. Baurmann[8] suggests a dynamometric method of diagnosis.

CASES

Group I. True Pulsating Exophthalmos. Carotid-cavernous communication.

Case 1. L. H., a 71 year old white female presented herself at the Illinois Eye and Ear Infirmary, service of Doctor L. Hoffman, on November 20, 1938. She had awakened one morning five weeks earlier with a peculiar pounding sensation in her head. There was no paralysis or weakness. The pounding noise continued. Two weeks later the right upper lid began to droop and both right lids became swollen. The head noise and swelling persisted. There had been no trauma of any kind.

Vision was 20/200 right and 20/70 left, corrected.

External examination of the *right eye:* The eye was proptosed 10 mm. The upper lid was ptosed and immobile. The palpebral fissure was two millimeters wide, compared to 11 mm. on the left, on looking upward. The veins of the upper lid were markedly engorged. The lower lid was completely everted by markedly chemotic palpebral conjunctiva. The globe was immobile and held in the primary position. Palpation of the globe through the upper lid revealed a thrill and pulsation synchronous with the radial pulse. The bulbar conjunctiva was somewhat chemotic, with dilated, tortuous episcleral veins. The cornea showed a 1½x8 mm. horizontal band of macerated epithelium in the interpalpebral fissure area and was insensitive to touch. The anterior chamber was slightly shallow. The pupil was 6.5 mm. in diameter, round, and

did not react to light or in convergence. The lens showed only a senile reflex with peripheral spoke opacities.

External examination of the left eye was essentially normal. The lens showed a senile reflex with cortical spoke-like opacities.

Fundus examination, right: The optic disc was of normal color with a slight edema of the margins. The veins were all engorged and tortuous, and showed variation in the size of their lumina in different areas. The arteries showed increased width of their reflexes. The veins were markedly compressed at the arteriovenous crossing. There was one flame-shaped hemorrhage lying just nasal to the disc. The macula appeared normal. Left fundus examination revealed no pathology other than arteriosclerotic arteriovenous nicking.

The peripheral visual fields were normal. The right blind spot showed slight enlargement concentrically. The left blind spot was normal in size.

On auscultation directly over the forehead a loud swishing murmur was audible, most marked on the right side. This disappeared on digital compression of the right common carotid artery. The right cornea was anesthetic. The extraorbital cranial nerves showed no abnormalities of function. Heart and lungs were normal. Blood pressure was 170 mm. Hg systolic, and 90 mm. diastolic. Superficial and deep tendon reflexes were normal.

Blood and urine studies were negative.

X-rays of the skull and orbits were entirely negative.

On November 25 a small conjunctival hemorrhage appeared on the lower nasal bulbar conjunctiva of the left eye, associated with increasing chemosis of the left lower palpebral and bulbar conjunctivae. On the 29th of November the exophthalmometer readings showed a 2 mm. decrease of the exophthalmos on the right and a 6 mm. increase on the left.

Fundus examination revealed no change in either eye from the previous findings. The right vision was the same; the left 20/100 corrected. The change was believed to be due to rearrangement of the venous circulation in the intercavernous sinuses.

On December 1, the patient became very drowsy and could not be aroused. Neurologically, no findings were present. The coma became deeper and the patient died within a few hours.

Post-mortem examination by Doctor Otto Saphir revealed the following findings relative to the brain:

"At the base of the brain an abnormality of the posterior cerebral artery is noted. This artery is seen to originate from the basilar artery first as a very delicate and small branch and forming the posterior communicans artery. From the region of the infundibulum much blood-tinged fluid exudes. The lateral ventricles are partially filled with bloody fluid. After removal of the corpus callosum, there was found a blood clot in the third ventricle, filling the whole lumen. The cerebellum reveals on section a large dark area, in which the normal substance is destroyed. This area is present in all lobes of the cerebellum and is seen to be connected with the fourth ventricle and the aqueduct, both of which are filled with clotted blood. The arteries of the base of the brain are filled with blood clots which continue in the more peripheral branches of the cerebral and cerebellar vessels. In addition, their intima contains many yellowish areas scattered throughout. The right cavernous sinus is markedly dilated. There is a communication between the internal carotid artery and the dilated sinus. At one point the latter is ruptured. The left cavernous sinus shows no changes. The region of the hypophysis on both sides and the region of the cavernous sinus are infiltrated with blood. There is clotted blood about the sella turcica."

Case 2. V. S., a 48 year old white female, entered the Illinois Eye and Ear Infirmary on August 22, 1938, on the service of Doctor S. J. Meyer. Two years previously she had injured the back of her head by a fall down a flight of stairs. She was unconscious for fourteen days following the accident. When she recovered consciousness she noticed a peculiar whirring noise in her head and prominence of the blood vessels of the right eye. The noise and prominent vessels persisted to the time of admission to the clinic. With any exertion or on stooping, the noise became louder and the patient experienced vertigo. X-rays of the skull taken at Cook County Hospital following the accident were negative for fracture.

Vision, corrected, was 20/20 with each eye.

The right eye was proptosed 5 mm. The lids were normal.

The right bulbar conjunctiva contained medusa-head-like, dilated, tortuous veins. There was no chemosis. The palpebral conjunctiva, sclera and cornea were normal. The corneal sensitivity was intact. The anterior segment of the globe was normal. The disc margins and color were normal. The retinal veins were dilated to about twice their normal size. The macula was normal in appearance.

Visual fields, both peripheral and central, were entirely negative.

General physical and neurological examination revealed only a bruit over the skull, most marked in the right frontal region. The blood pressure was 120/80. X-ray examinations of the skull and orbits showed no pertinent findings.

Blood and urine studies were negative.

Two unsuccessful attempts were made in September, 1938, by Doctors C. A. Neymann and R. W. McNealy, to visualize the vascular lesion by X-rays. Twenty-five cc. of thorotrast were injected intra-arterially at each attempt.

On October 26th the intraocular tension was 35 mm. Hg. right and 22 left. One drop of 2% pilocarpine was ordered four times daily in the right eye. On December 14, 1938, the tension was 35 mm. right and 32 mm. left. Pilocarpine was then advised for use five times daily in both eyes. On December 21st the tension was 31 mm. right and 29 left.

A cervical truss was then made and used for compression of the right common carotid. This was used four times daily, beginning with ten minute periods, and increasing to about a half hour. No symptoms were produced by the compression. The bruit usually disappeared with compression, although occasionally it could then be heard in the left ear. The compression was continued until ligation was performed on March 9, 1939, by Doctor C. H. Christoph. After careful dissection down to the common carotid, the artery was tied with heavy braided silk. Part of the omohyoid muscle was tied under the suture to prevent cutting into the arterial wall. Following the ligation the bruit ceased. Recovery was uneventful, except for paralysis of the right vocal cord, which was due to surgical involvement of the right vagus nerve. Examination of the patient on March 25 revealed 20/20 corrected vision in each eye. Tonometric tension was 19 in each eye. The right exophthalmos measured 3 mm. There was no change in the appearance of the conjunctival or retinal veins. No bruit could be heard. The patient stated that when she exerted herself she sometimes heard a noise in the left ear. On June 21, 1939, the patient noticed the appearance of a mass beneath the incision area in the neck. She was seen a week later when a diagnosis of aneurysm of the common carotid artery was made and the patient referred back to the otolaryngological service. On July 1, 1939, under local anesthesia the neck was re-opened by Doctor Lindon Seed, the neck surgery consul-

tant, and a golf ball sized sac exposed. It was adherent to the surrounding tissues.

It was tied off above and below with braided silk. The sac was opened and an opening found in the posterior medial wall of the common carotid artery. This was closed and the aneurysmal sac removed. In the opinion of Doctor Seed the aneurysm was a sacular affair from perforation of the wall of the carotid, probably by the previously placed suture. There was no connection with the internal jugular vein. The inferior thyroid, lingual and external carotid arteries were ligated with catgut. The internal jugular vein was ligated and cut at its lower end.

Following the operation a Horner's syndrome developed due to paralysis of the sympathetic nerve on the right. On July 25, 1939, the right palpebral fissure was 3 mm. less in width than the left. The right pupil was 2.5 mm. in diameter and the left 4 mm. in diameter. Both reacted briskly to light. The right exophthalmos measured 3 mm. The tonometric tension was 21 mm. Hg in each eye. Visual fields and fundus examination showed no change from original findings.

In retrospect it is seen that in spite of the re-establishment of circulation following the first ligation, there was relief from the bruit and normalization of intraocular tension. This speaks for the adequacy of partial ligation.

The complication of aneurysmal sac formation as a result of perforation of the arterial wall is extremely unusual and is one of the rarest of complications. This case also showed two other complications, which may occur in this type of surgery, namely laryngeal nerve paralysis and Horner's syndrome.

Case 3. F. T., a 59 year old white male, was first seen at the Illinois Eye and Ear Infirmary on July 21, 1937. On June 10, 1936, he was struck on the left parietal region with a blackjack during a robbery. There was no unconsciousness. The left eye protruded, and the patient had severe left headache and a pounding noise in the ears, synchronous with the radial pulse. On June 30, 1936, a subconjunctival hemorrhage and marked chemosis occurred in the left eye. He was hospitalized in July, 1936, and the left conjunctiva was incised to remove some of the chemotic fluid. Four weeks later the right eye became prominent and the right conjunctiva chemotic. In August, 1936, the patient experienced diplopia which persisted. A portion of the right chemotic conjunctiva was ex-

cised in October, 1936. At the time of admission to the clinic the patient desired treatment for his right lower lid.

Vision was 20/20 in each eye corrected. Tension was 23 mm. each eye, with the Gradle-Schiotz tonometer.

There was marked ectropion of the right lower lid with exposed, red, rough conjunctiva. A symblepharon was present in the lower fornix. Exophthalmometer readings were 23.5 mm. right and 21 mm. left. A right esotropia of 10° and right hypertropia of 8° were present. Excursions of the extraocular muscles in the six cardinal directions of gaze indicated pareses of the the right external rectus, the left superior rectus and the left inferior oblique.

The right bulbar conjunctiva contained many large dilated veins. The anterior segment of the globe was entirely normal. Ophthalmoscopically the right disc was normal. The veins were prominent and the macula normal.

The left bulbar conjunctiva showed engorged veins as on the right. The anterior segment was normal. The fundus examination revealed slight elevation of the left disc. There were eight or ten small hemorrhages along the peripheral course of the superior and inferior temporal veins. The macula was normal.

Visual field examinations were entirely normal.

Physical examination revealed a loud bruit, synchronous with the pulse, over the orbits and temporal regions. Blood pressure was 140/84. Blood Wassermann and urine were negative.

A diagnosis of bilateral pulsating exophthalmos due to arteriovenous aneurysm in the cavernous sinus was made. On compression of the common carotid on the left, the bruit disappeared, but the patient was unable to talk and experienced a tingling sensation in the opposite arm and hand. For this reason operation was believed contraindicated.

On November 21, 1938, the vision was 20/20 each eye. During the past nine months the bruit had decreased markedly and no longer annoyed the patient. The diplopia persisted. The right pupil was half a millimeter larger than the left. The conjunctival vessels were much less prominent. The exophthalmometer readings were 22 mm. right and 21 mm. left. The bruit was still audible and disappeared on carotid compression.

With 20 prism diopters, base out, and 15 prism diopters, base up, in front of the left eye the patient felt very comfortable and no longer experienced diplopia. A 26 prism diopter prism at axis

21 was then ordered as a slip over for the left eye. This was made up by J. C. Copeland of the Riggs Optical Company in the form of a plano-meniscus prism with 6.00 D. base curves and cemented to the slipover lens. The patient had single binocular vision with good depth perception with the lens and experienced no chromatic aberrative sensations after the first day of its use.

This case is an example of spontaneous improvement in a case of bilateral pulsating exophthalmos where operation had been previously considered, but not done because of the results of digital compression. The visual result with prisms was excellent.

Case 4.* W. McK., a forty-four year old white female, was seen by Doctor Harry S. Gradle on February 24, 1939. Two months previously she had noticed a buzzing noise in her left ear which persisted. Three weeks previously the left eyelids and conjunctiva had become swollen. In the summer of 1938 she had bumped the left side of her head against the instrument board of an automobile when it stopped suddenly. On examination at that time by Doctor Grant H. Laing, he found that the left pupil was dilated, whereas at a previous physical examination on October 14, 1937, the right pupil had been slightly larger than the left but both had reacted normally to light.

Vision was 20/20 corrected each eye. Tonometric tension (Schiotz) was 20 mm. Hg each eye.

The right eye was entirely normal.

Left exophthalmos of 3 mm. was present.

A loud bruit was heard over the orbits, particularly on the left side, and also in the left temporal region. Digital compression of the left common carotid caused cessation of the bruit and the subjective noise. The left upper lid was edematous, particularly near the inner canthus. The outer half of the bulbar conjunctiva was edematous. The sclera was mildly injected. There was slight limitation of motion of the eye outward and possibly downward. The anterior segment was entirely normal. The inferior border of the left disc was somewhat hazy. The veins were slightly engorged and somewhat tortuous. The arteries were a little fuller than normal but not tortuous. The remainder of the fundus was normal.

Peripheral and central field examinations were entirely normal.

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* The authors wish to thank Doctors H. S. Gradle, E. Oldberg and G. H. Laing for permission to use this case.

A diagnosis of carotid-cavernous aneurysm was made and the patient given the cervical collar previously described (Case 2) for prolonged compression of the common carotid artery.

Group I. True Pulsating Exophthalmos. Orbit arteriovenous aneurysm.

Case 5. J. C., aged nine, entered the hospital on October 29, 1938, on the service of Doctor S. J. Meyer. Three weeks previously he had been struck between the right globe and lateral orbital margin by one prong of a pitchfork which was thrown from a haystack. The eye swelled shut immediately following the injury. The blood which had suffused under the skin of the upper lid had largely disappeared by the time the patient was admitted to the hospital.

On examination the right vision was 20/20 each eye, corrected. Tonometric tension with the Schiotz tonometer was 26 mm. Hg right, and 18 mm. left.

The right upper lid showed slight bluish discoloration, some swelling and dilated subcutaneous veins. There was 7 mm. exophthalmos on the right. The right palpebral fissure was 8 mm. wide, while the left was 10. There was no tenderness on pressure over the right orbital margin. A thrill was palpable and systolic bruit audible over the upper lid area on the right. Digital compression of the right common carotid in the neck stopped the bruit and thrill.

The right bulbar conjunctiva contained markedly dilated and tortuous veins. The anterior segment was otherwise entirely normal by gross and slit-lamp examination. The corneal reflex was intact. Ophthalmoscopically the disc margins were slightly blurred. The arteries were normal in size. The veins were tortuous and dilated to about twice their normal size. The macula was normal in appearance. The left eye was entirely normal.

No diplopia could be elicited by the red glass test. Depth perception was normal, as tested with Guibor cards and stereoscope.

Peripheral and central visual field studies were entirely normal. X-ray examination of the right orbit to rule out possible fracture of the posterior wall or penetration by the pitchfork was negative.

Blood and urine studies were entirely negative.

Neurological examination by Doctor B. Boshes showed no evidence of any nerve involvement which might point to involvement of the cavernous sinus.

Diagnosis of traumatic arteriovenous aneurysm of the right orbit was made. In view of the recency of the trauma it was decided to wait for some time longer to get more vascular equilibrium in the orbit before attempting a surgical ligation of the venous part of the fistula in the orbit. Several attempts to get the child to return for surgery, however, were unsuccessful.

Group II. Pseudopulsating Exophthalmos. Orbital angioma.

Case 6. S. K., a 34 year old white female presented herself at the Illinois Eye and Ear Infirmary, service of Doctor P. O'Connor, on January 31, for surgical treatment of an irradiation cataract. Ten years previously she had received a series of irradiation treatments for a hemangioma of the left orbit. For the past nine years she had been unable to see with the left eye and one year ago had noticed a white spot in her left pupil.

Vision with the right eye was 20/20 and Jaeger I uncorrected. Left vision was handmovements at one foot. Tension with Schiotz tonometer, using Friedenwald's corrected values, was 22 mm. Hg right and 23 mm. Hg left.

The left lids and periorbital skin showed evidence of irradiation and thrombosis of skin vessels. The left eye showed 4.5 mm. exophthalmos. The right palpebral fissure was 10 mm. wide and the left 11 mm. on looking upward. The left eye was visibly and tactilely pulsating synchronously with the pulse. No bruit was heard.

The left lens was extracted by the extracapsular method on February 1, 1939. Recovery was uneventful. On February 12, the left fundus was examined, and found to be entirely normal.

TREATMENT AND RESULTS IN CAROTID-CAVERNOUS ANEURYSM CASES

Beneficial results of treatment whether surgical or nonsurgical, depend on two factors: the first, the prevention of short-circuiting of blood into the venous system, and second, decrease of intracranial pressure for a sufficient length of time to promote thrombosis. Dandy has emphasized the second factor.

Rest as a means of promoting clot formation is particularly important early and especially in the traumatic cases.

Drugs which stimulate blood coagulation have been used by many authors. Those most used have been calcium salts and gelatin solutions. The latter, advocated in warm 2% solution by Paulesco[9],

is injected subcutaneously in doses of 100 to 250 cc. every four to eight days for a total of ten to twenty-five injections.

It is probably wise to continue conservative treatment until vascular compensation has occured. Of course, in severe trauma with bleeding from the nasal orifices, ligation may have to be done immediately. If the average case is left untreated, the vision may be lost or thrombosis of the cavernous sinus with extension of the thrombus to the other venous sinuses may occur.

The only disadvantage of obstruction of the proximal segment of the artery is that the blood supply of the brain is thereby decreased, at times sufficiently to cause hemiplegia or even death. For this reason, every ligation should be preceded by compression to determine the adequacy of the anastomotic cerebral blood supply.

Compression may be digital or by a cervical truss. Digital compression is performed by pressing the common carotid artery against the tubercle of the sixth cervical vertebra for gradually increasing periods of time three to six or eight times daily, beginning with one minute and increasing to about forty-five minutes for each period. By use of a cervical truss alone, one of the authors' collected cases was cured and three were improved.

There are two avenues of approach to ligation, arterial and venous. The arterial may be extra- or intracranial. The extracranial consists of ligation of the internal and the common carotid arteries or the latter's branches, singly, simultaneously, or consecutively, or mechanically plugging the internal carotid artery with either long strips of muscle (Brooks) or with paraffin inserted at the origin of the artery in the neck. The intracranial approach is that of Dandy, recommended after failure of the extracranial ligation.

The ligation may be partial or total. For the partial type many mechanical means have been employed, including clamps and fascia lata. Dorrance[10], in two splendid papers, objected to the use of clamps since they may injure the vascular endothelium and contribute to the danger of embolism.

The choice of whether to ligate the common or internal carotid is not a settled question. In Dorrance's series of 275 cases ligation of the internal and common carotids closely approximated each other, with slightly better results with the common carotid ligation. A few more recurrences occurred after the internal carotid ligation.

In the authors' series, 34 cases had ligation of the common carotid alone. Seventeen (50%) were cured, nine were improved six failed, one died and seven recurred.

Thirty had ligation of the internal carotid alone. Fifteen (50%) were cured, six improved (two had hemiplegia), seven failed and one died. Three recurred, and the result in one case was not stated.

These results indicate no advantage of one over the other.

It has been shown by Blackman,[11] Dorrance,[10] Matas,[12] and many others that after ligation of the common carotid there is a reversal of blood flow in the external carotid. Almost half of this recurrent flow, according to Dorrance, comes from the superior thyroid artery. Part of it comes from anastomoses between the descending branches of the occipital and the deep cervical branches of the costovertebral trunk, the lingual, facial, occipital and temporal with the corresponding arteries of the opposite side. Dorrance believes this reverse flow to be important in preventing both cerebral anemia and the reversal of flow in the distal segment of the artery which would occur if the internal carotid were ligated.

In spite of the argument of greater security in ligation of the common carotid, it would appear to the casual observer that the restoration of blood flow through the internal carotid by collateral circulation is just the effect we are trying to overcome. Actually, when the internal carotid itself is ligated, the reversed flow through the aneurysmal opening from the circle of Willis tends similarly to continue the syndrome of pulsating exophthalmos. Since there is a flow of blood through the aneurysmal communication in either case there seems to be little mechanical advantage of ligation of the common carotid over the internal, or vice-versa. To the authors it appears that the added safety of common carotid ligation makes it the better choice.

On the venous side the surgical approach consists of ligation or extirpation of the ophthalmic veins, use of diathermic coagulation of the veins (Fiolatov[13]), injection of sclerogenic solutions and direct ligation of the veins after enucleation. These act by causing thrombosis in the venous channel, extending to the fistula region.

For the orbital arteriovenous communications the vessels may be approached trans-conjunctivally between the globe and the lateral orbital walls (Wheeler) or by the Kroenlein operation or one of its modifications.

From the results observed by the authors in this series, they advocate, first, rest and compression, meanwhile watching the vision and general condition. Then, partial or total ligation of the common carotid artery. Later, the superior thyroid or external carotid can be dealt with if necessary.

Spaeth,[14] in his recent book on ophthalmic surgery, points out that roentgenologists have obtained satisfactory results with deep X-ray therapy in carotid-cavernous cases. This method has, as yet, had insufficient trial. Inability to limit the area of reaction to the desired small area, and the possibility of extension of the thrombotic process as a result of irradiation, seem to be reasonable points against its use.

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DISCUSSION

F. Bruce Fralick, M.D., Ann Arbor, Mich.: Doctors Meyer and Sugar have done well in presenting this interesting discussion on Pulsating Exophthalmos. They have left very little to be added to this subject in the light of our present knowledge concerning it. To the six cases of pulsating exophthalmos reported by the authors, I wish to add the summaries of eight more, previously unreported cases of the same condition seen between the years 1924-1939 in the University Hospital, Ann Arbor.

Case 1 — G. S., age 28, December 26, 1924

History: During a fight received a blow on the back of his head and was unconscious for five days. The right eye was swollen since time of injury.

Findings: Right eye was proptosed and pulsated on palpation. (Amount of proptosis was not recorded.) There was bruit over right side of head. Patient was deaf in right ear.

Diagnosis: Arteriovenous aneurysm of right internal carotid in basilar sinus. Pulsating exophthalmos O.D. On December 29, 1924, right internal carotid was ligated, followed by cessation of bruit and pulsation. The following day a slight bruit was heard. Patient discharged and never returned.

CASE 2 — J. A., age 62, May 12, 1929

History: Two months previously fell from street car and struck back of head which was followed by period of unconsciousness which lasted three days. Two months later heard a roaring in head which seemed to radiate in the form of a thrill and pulsation to left eye. Constant pain in left orbit, worse when in prone position.

Findings: V. w.c. 6/15 O.D., 6/60 O.S. Intraocular tension normal. Edema of upper and lower lids. Chemosis of conjunctiva O.S. with marked engorgement of conjunctival and episcleral veins. Pulsating exophthalmos O.S. measuring 28 cms. and 19 cms. O.D.

Fundus: Negative O.D. O.S. shows arterial and venous engorgement and slight edema of the disc. X-ray of the skull was negative. Compression of common carotid gave no general disturbances and ligation of common carotid was performed which resulted in cessation of pulsation in the left orbit.

The bruit was disappearing and was only faintly heard when the patient was last seen two months later. There was much improvement from the proptosis, chemosis and edema of the lids. Wasserman was four plus.

CASE 3 — R. Z., age 55, March 17, 1930

History: Five months previously was in auto accident followed by unconsciousness for three days. On regaining consciousness he was blind in both eyes and was conscious of a thumping along the right side of head just in front of the ear. Vision started to return one week later.

Examination: V. 6/30 O.D. 6/10 O.S. Tension normal. Exophthalmometer readings 22 O.D., 20 O.S. Diplopia in all fields. Complete paralysis of right externus, partial parlaysis of left externus. Slight chemosis and hyperemia of bulbar conjunctiva. Direct and accommodative reflexes absent O.D. Right fundus shows disc edematous and hyperemic. Veins engorged and tortuous. Marked engorgement of small capillaries over and about the disc. Left fundus negative. Bruit synchronous with pulse over right side of head and orbit. Day following admission patient had a severe epileptiform seizure following which roaring in head was almost gone. Bruit was not heard, so it was thought that patient had a spontaneous thrombosis of the aneurysm. Cause of convulsion thought to be temporary cerebral anemia. He was confused for several days but this cleared up completely. Kahn four plus.

Diagnosis: Arteriovenous aneurysm of the right internal carotid artery in the cavernous sinus. Pulsating exophthalmos O.D.

Course: Returned one month later with right convergent strabismus and proptosis O.D. Slight bruit over the right side, and complained of peculiar noises in the right temporal region. Patient did not feel that his symptoms were sufficient to warrant surgery since ligation of the carotid was advised.

CASE 4 — E. E., age 64, June 3, 1930

History: Two months previously patient was taken with a vomiting attack which lasted off and on for two days, during which she had a sudden pain in right side of head which did not leave for two months. This pain was accompanied by swelling of right eyelids, but no visual loss except that due to the ptosis which developed suddenly with the onset of the pain. Patient noticed a roaring of the ears for past ten years, which has been much worse since the onset of the present pain.

Findings: V. without correction 6/20 O.D., 6/30 O.S. Complete ptosis of right upper lid with marked proptosis O.D. Exophthalmometer readings 19 O.D. 12 O.S. Edema of lids O.D. accompanied by chemosis of bulbar conjunctiva, more marked above than below. Conjunctival veins were dilated and tortuous. Right globe is fixed. Complete paralysis Nerve III, IV and VI. There is marked thrill and pulsation over right eyeball and temporally which is synchronous with the pulse. Loud blowing murmur heard throughout the entire region. Intraocular tension normal. Fundus showed nerve head edematous and hyperemic; veins engorged and tortuous. Edema of retina. X-ray of skull negative.

Diagnosis: Pulsating exophthalmos. Arteriovenous aneurysm — site undetermined. General condition too poor for surgery. Patient lost sight before intermittent carotid pressure could be instituted.

CASE 5 — W. K., age 18, April 21, 1937

History: Six months ago patient received a charge of shot from shot gun at two feet distance, shot striking face, rupturing left globe and one shot perforated the right globe.

One month after injury tissues of left orbit became swollen and pulsated. Patient states that arteriovenous aneurysm of orbit was removed, two months after injury which recurred soon thereafter.

Findings: V. moving objects O.D., O.S. enucleated. Tension normal O.D. The orbital tissues on the left were edematous and conjunctiva chemotic. A pulsating arteriovenous aneurysm could be felt in the orbit and there was a bruit and thrill present through the upper lid, forehead and temporal regions. Pulsation was synchronous with pulse. A distinct bruit could be heard in the carotid region of the neck. Compression of the common carotid produced no undue symptoms so in April, 1927, the internal and external right carotid were ligated with disappearance of symptoms. Bruit, pulsation and thrill returned early in 1931, but the patient no longer has roaring sensation in his head. The orbital tissues were opened on June 5, 1931, and all large vessels ligated. It was impossible to tell which arteries and veins were involved. Permanent relief until lost sight four years later.

CASE 6 — J. B., age 11, December 13, 1937

History: In June, 1934, was struck just below the medial end of the right supraorbital ridge with a pitch fork tine. In a day or two the eye began to protrude and has been so since. In the summer of 1937, the swelling has been extending below the eyes and up into the forehead with palpable pulsation.

Examination: V. 6/30 O.D., 6/9 O.S. Tension normal O.U. Aneurysmal dilatation of the supraorbital veins O.U. and also communicating vessels across the bridge of the nose. Right eye is displaced downward 1 cm. and proptosed 20 mm. O.D., whereas the left is 15. Globe pulsates on palpation, the pulsation being synchronous with heart beat. Loud bruit heard all over right side of head and less on left. E. O. M. are limited on right in all directions. Veins on right fundus are engorged and normal in left.

Diagnosis: Pulsating exophthalmos O.D. Arteriovenous aneurysm right orbit.

Operation: December 17, 1937, brow incision. Number of large veins were found and ligated but no true arteriovenous aneurysm as such could be discovered. Pulsation and bruit disappeared.

Last seen — five months later. Exophthalmometer readings 15 O.D., 14 O.S. No bruit or pulsation. Venous engorgement in right fundus disappeared.

CASE 7 — L. B., age 15, December 16, 1938

History: At age 13, prong of pitch fork entered left orbit through the inner end of the upper eyelid. Normal healing, but left a boggy swelling in this area.

Findings: V. w.c. 6/6 O.U. Intraocular tension was normal. Exophthalmometer readings O.D. 16, O.S. 20. Palpebral fissures measure vertically O.S. 9, O.D. 11. Poor adduction O.S. and complete paralysis of external rectus O.S. Pulsation of left eye and of the medial half of the left upper eyelid where there is a boggy fullness. Pulsation is synchronous with heart beat together with thrill and bruit. Bulbar conjunctival veins engorged. Fundus examination negative except for venous pulsation on disc O.S.

Diagnosis: Traumatic arteriovenous aneurysm — left orbit. Paresis left external rectus.

Operation: Excision of arteriovenous aneurysm involving the ophthalmic artery and vein.

Recurrence: None after two years.

CASE 8 — B. D., age 37, May 4, 1939

History: No complaints until the previous March when she developed influenza. A few days after the onset of the influenza the left eye became swollen and sore, tender to pressure; symptoms worse in A.M., decreased as the day progressed.

Findings: V. 6/12 O.U. Tension normal O.U. Exophthalmometer readings 16 O.D., 22 O.S. There is a slight ptosis O.S. Paresis of internal rectus O.S. Conjunctival and episcleral vessels were dilated and tortuous. Fundus shows pulsation of vessels on the disc., O.S. Veins were dark and engorged. Pulsation of globe was faintly visible and a bruit was heard over left side of the head, but to a less marked degree after carotid pressure.

Impression: Arteriovenous aneurysm of cavernous sinus.

Course: Patient was put on intermittent compression over the carotid with no apparent relief, even though compression was increased up to two

hours daily. On July 1, 1939, ligation of left internal carotid was done with complete disappearance of the bruit and pulsation. When seen two months later there was still a bruit present over the left eyeball; no pulsation.

That this condition is rarely seen is illustrated by the fact that the eight cases added in this discussion represent a frequency of one case of pulsating exophthalmos to every 22,500 patients seen in the clinic.

The observation of Kattan that in the presence of craotid-cavernous aneurysm the vision may remain normal for over twenty-five years even when accompanied by rather marked engorgement of the retinal vessels, illustrates very nicely Whitnall's findings in regard to the venous drainage of the eyeball and orbit. He states that the central vein of the retina opens most frequently separately into the cavernous sinus, rarely into the superior or even inferior ophthalmic vein, but that it always has at least one side connection, commonly with the superior ophthalmic vein. This anastomosis is not only important in carotid-cavernous aneurysms but explains why we do not have the clinical picture of occlusion of the central retinal vein in cavernous sinus thrombosis.

For the sake of completeness of the discussion of pulsating exopthalmos a little more might be said in regard to pulsation of the eyeball associated with defects in the wall of the orbit as pointed out by the late John Wheeler.* He stated that these bony defects may be due to (a) a congenital arrest of development, (b) an accompaniment of plexiform neuro-fibromatosis of the orbit (von Recklinghausen's disease), and that in this condition the bony defect may be congenital, (c) or the result of surgical removal of the orbital roof as in the Nafzinger operation for malignant exophthalmos. Wheeler also pointed out certain important differential diagnostic points between arterio-venous communications and orbito-cranial communications which we should keep in mind when a case of pulsating exophthalmos is presented. In orbito-cranial communication, the pulsation is more evident to the examiner on direct observation while in arterio-venous communication the pulsation is only seldom seen but may be elicited by compression of the globe and orbital tissues. The pulsation is synchronous with the pulse in both conditions. The pulsation is not accompanied by the annoying bruit as is always the case in arterio-venous communication. There is an absence of the markedly dilated veins of the eyeball, lids and surrounding tissues as seen in arterio-venous communications.

All the diagnostic aids possible must be used to differentiate the types and location of the lesions causing pulsating exophthalmos if we are to obtain the highest percentage of good results for our patients.

Edmund B. Spaeth, M.D., Philadelphia: In any presentation on any subject which is as exhaustive as is this, one could select any one of many different subdivisions of the subject, or points of interest which are developed, for a set discussion. Questions on anatomy, on the etiology and the mechanics of the condition, the rational of the therapy utilized, and the

* Bull. Neural. Inst., New York, 5:476-484, Aug., 1936.

gross local as well as the gross distant pathology, all are of equal importance with that which I would like to emphasize, namely differential diagnosis. Even this is to be further limited in that only those cases are being considered for differentiation which have one or more of the common symptoms of, a history of trauma, a bruit — or at least a pounding or throbbing sensation, exophthalmos, definite visable pulsation, and modification of the signs and symptoms by temporary pressure upon the centripetal blood vessels, or by changes in the position of the patient's head. In the final analysis, when considering vascular conditions alone, the biggest single diagnostic necessity is the differentiation of an orbital, i.e., arterio-venous aneurysm from an intracranial aneurysm of one of the larger intracranial cerebral blood vessels. Moller, who studied 555 cases of this later condition, stated that in none of these was choked disk found, bulbar protrusion was rare, oculo-motor paralyses were quite common, and true pulsation was never seen. The author's sub-groups, as they arranged them, are proper and clear.

With this in mind, six cases are to be very briefly presented — none of which was a pure case of carotid-cavernous sinus nor carotid-jugular bulb aneurysm, but all of which illustrate possibilities and complexities demanding for themselves correct differential diagnosis and the proper therapy.

Case 1. Colored boy, direct local trauma from a base ball two years before; slight exophthalmos of the right eye; no bruit, pulsation of a mass in the lower lid and in the lower cul-de-sac, recent increase in the size of the mass; no fundus changes; at times brief periods of a rather marked edema of the lower lid and the palpebral conjunctiva. Operation and removal of a cavernous hemangioma extending posteriorly into the orbit over the edge of the bony orbit.

Case 2. Young man, age 16, deep stab wound of the orbit resulting in an almost complete external ophthalmoplegia; exophthalmos and edema of the conjunctiva which was immediate in its development; emphysema, and a deep pounding sensation deep in the orbit; no fundus changes. Spontaneous recovery except for the ophthalmoplegia. Some residual exophthalmos still present. (This case may still develop a true pulsating exophthalmos.) And incidentally this case may still develop an arteriovenous aneurysm.

Case 3. Male, age 40. Pounding migraine like headaches with nausea and with vomiting; no bruit; partial external ophthalmoplegia, third nerve, left eye; very slight exophthalmos right eye; no edema of the conjunctiva; bilateral field defects tending toward a binasal hemianopia, with an increase in the field defects by reason of right external carotid pressure but with a definite amelioration of symptoms at the same time; bilateral high degree of haemorrhagic form of choked disks, first appearing in the right eye, several days later developing to almost the same degree in the left eye. No disturbances with the sense of smell. The pre-operative diagnosis was that of a space taking lesion near or at the optic foramen on the right. Operation revealed a bilateral aneurysmal varix of high degree on the right side, less so on the left side, of the anterior cerebral arteries. These vessels were ligated carefully. Complete recovery without further symptoms of any

kind, now for over three years. That case following ligation has made an uneventful recovery with complete cessation of symptoms.

Case 4. Male baby, age 12 months. Difficult birth with forceps delivery; the early development of a lesion above the right supra-orbital ridge which was elevated, covered with wrinkled skin and with a definite pulsation which could be modified decidedly by the pendent or erect position of the infant's head; unilateral exophthalmos on right; partial or incomplete external ophthalmoplegia; edema of the conjunctiva of the right eye, normal fundus picture on left, right fundus could not be seen because of local inflammatory changes; x-ray showed a round bony defect in the frontal bone immediately above the supra-orbital ridge. Operation, median tarsorrhaphy because of the exophthalmos and the lagophthalmos, exploration of the elevated mass, diagnosis meningo-encephalocele through congenital dehiscence or birth-trauma defect in the frontal bone.

Case 5. Male, age 24. Direct blow to the right eye from a broom stick which had been thrown as one would cast a spear, the patient having been struck by the tip of the broom handle. Rupture of the choroid and the retina with secondary optic nerve atrophy, exophthalmos, pulsation with a bruit, partial or incomplete external ophthalmoplegia. Fractional carotid ligation, completed, resulted in a partial correction of the bruit and a complete correction of the pulsation. The exophthalmos continued to increase. Enucleation was done for an exploration of the orbit and resulted in a terrific haemorrhage which proved to be from a dissecting form of aneurysm arising from a venous sinus and passing through the sphenoidal fissure into the orbit, possibly an extension from the original internal carotid-cavernous sinus aneurysm, or even more likely, internal carotid-jugular bulb aneurysm which had increased in size and extent from venous blood stream back pressure following the external artery ligations. Uneventful recovery after immediate haemostasis and subsequent roentgen therapy.

Case 6. Girl, age 12. Neurofibromatosis of the skin of the upper and the lower lids since birth. Right exophthalmos with pulsation of the eyeball. Mass felt deep in the orbit which was movable and which did not seem, of itself, to pulsate. Gross defect in the right zygomatic arch. Encapsulated sequestrum, painless and without inflammatory reaction (x-ray diagnosis) in the outer wall of the orbit. No oculo-motor disturbances; incidentally, that was x-ray diagnosis only; visual acuity 6/30. Bruit could not be heard by the examiner and was not appreciated by the patient. The pulsations of the orbital mass were affected but little, if at all, by pressure upon the carotids. Orbital exploration through a Kroenlein incision and partial orbital wall resection revealed an intraorbital neurofibroma which was removed simultaneously with the tumor mass of the same tissue from the upper and the lower lids and also from the deformed zygomatic fossa. Removal of the encapsulated sequestrum at the same time. A large globular mass was then uncovered in the orbit, entering probably through the sphenoidal fissure, compressible, and with a clear-cut, definite pulsation, this applied to the overlying eyeball with quite a sharp impact. Operative wound closed and the patient placed upon deep roentgen therapy by a radiologist at her home town. After two years intermittent but extensive x-ray

treatment there has been an almost complete recession of the exophthalmos and a complete disappearance of the pulsation. Two years after the initial surgery, case was completed from a cosmetic standpoint with the correction of a slight degree of ptosis and with a canthoplasty for a minor post-operative defect which existed at her outer canthal angle.

From this very brief presentation of six not too unusual cases it seems as if differential diagnosis is a not unimportant factor in many cases presenting a major number of those signs and symptoms, already detailed by the authors, which suggest pulsating exophthalmos due to internal carotid-cavernous sinus or jugular bulb aneurysm.

T. L. Terry, M.D., Boston: In 1934 Mysel and I reported a case of pulsating exophthalmos resulting from an abnormal anterio-venous communication in the neck between the internal jugular and the internal carotid artery. Since that report was made there have been several developments. The pulsating exophthalmos has recurred as Reed predicted in his discussion of the original report. This, Reed based on the spontaneous onset attributable to congenital defects and such congenital defects are usually multiple. The patient had resumed heavy labor as a hod-carrier. The X-ray localization as reported by means of thoroium dioxide proved faulty in that another patient with an arterio-venous communication in the cavernous sinus showed a similar shadow in the neck with the use of the same technique. In spite of Dandy's criticism it seems most logical to believe that the abnormal communication, as originally reported, was located in the neck because whirlpools and eddies of arterial blood were seen in the internal jugular vein, a condition I have seen only near the location of arterio-venous communications since mixture of arterial and venous blood would be complete a relatively short distance central to the aneurysm. It must be remembered that the murmur was loudest in the upper part of the neck, was very slightly audible over the skull and the eye, and the amount of exophthalmos was never large. Although the pulsating exophthalmos has recurred, it is not as extensive as it was previous to the operation.

Localization of abnormal arterio-venous communications is important because the communication is not always intracranial. It may be intraorbital as shown by the case reported by Verhoeff and myself in 1938. In event the communication is not in the cavernous sinus, it may be removed. Based on the experience in several instances, the ultimate vision of the exophthalmic eye is often reduced. If that conclusion is correct, one should consider sacrificing the eye if by so doing the aneurysm can be eradicated. This is a surgical possibility if the aneurysm is in the orbit behind the globe. Verhoeff successfully obliterated the aneurysm causing a pulsating exophthalmos by enucleation and by ligation of the orbital vessels.

The abnormal arterio-venous communication can be visualized and thereby accurately located by the use of radiopaque substances injected into the artery. The correct technique for getting this result appears to be that used by Doctor J. C. White, a neurosurgeon of the Massachusetts General Hospital. Relatively recently he exposed the carotid in a patient with typical extensive pulsating exophthalmos and placed a band of fascia lata around the artery. The fascia lata was made to constrict the vessel by successively

stitching it tighter. After the carotid was occluded considerably and the flow of blood through it slowed down, some 10 cc. of thorium dioxide was injected rapidly into the vessel distal to the fascia lata and the X-ray film exposed simultaneously with the injection. Doctor White gave me the lantern slide shown here which demonstrates beautifully the arterio-venous aneurysm located in the cavernous sinus in this instance.

PHILIP MERIWETHER LEWIS, M.D., Memphis, Tenn.: I wish to add a short report of three cases of pulsating exophthalmus, from the Eye Service of the John Gaston Hospital, Memphis, Tennessee, to those presented by Doctors Meyer and Sugar.

1. A colored woman, age 61 years, had fallen and struck the left side of her head several months before symptoms began. There was no history of any other injury. She complained of a noise in her head as well as the protrusion of her eye. Exophthalmous was so marked that the eyelids failed to completely cover the globe. Ligation of the superior ophthalmic vein was performed without benefit. Ligation of the internal carotid was then performed without development of any untoward symptoms. The eye had to be enucleated because of a sloughing of the entire cornea. When last seen about six months after the operation she complained that the noise in her head had recurred but it could not be heard with a stethoscope.

2. A colored man, age 25 years, gave a history of two distinct head injuries. The first occurred about six weeks before the second, and consisted of a blow with a stick on his left eye and the left frontal region. This eye was removed elsewhere shortly after the injury, evidently on account of a ruptured globe. Ten days before we first saw him he was attacked by robbers and struck over the right eye with a blackjack. Almost immediately the right eye "popped out of its socket" so that he could not completely close the eyelids. The eye had begun to pain a few days later. He did not complain of a noise in his head.

Examination showed the eye greatly proptosed and the conjunctiva so edematous that it hung completely over the lower eyelid and rested on the cheek, giving an appearance similar to a large protruding hemorrhoid. The lower portion of the cornea was constantly exposed and had ulcerated. A gray infiltration of the parenchyma extended upwards from the ulcer over the pupillary area so that the vision was reduced to 20/100. All veins; conjunctival, scleral, retinal and those of the forehead were enormously dilated. The intraocular tension was not increased. A definite bruit could be heard over the frontal region with the unaided ear and with a stethoscope an aneurysmal hum could be heard over the entire skull. No thrill or vibration could be detected by palpation. The eye was immobile.

Ligation of the internal carotid was performed three days after he was first seen. No disturbance was caused by this procedure. Improvement followed immediately, the eye returned to its normal position and the veins became normal in size. The cornea healed, but the prolapsed conjunctiva remained without improvement. About three weeks later the prolapsed conjunctiva was sutured back in place with mattress sutures of heavy black silk. This eye would undoubtedly have been lost had not ligation of the internal carotid been promptly performed.

3. A colored man, age 41 years, gave a history of severe headache for two weeks before admission to the hospital. Six days previously he first noticed a noise in the left side of his head and the following day his left eye was swollen. The swelling greatly increased and a throbbing pain was present in his eye all the time. Pain was also present in his left face and upper teeth. He stated that he had never had any head injury and no previous eye trouble.

Physical examination was negative except for the ocular findings. The eye was greatly proptosed so that the entire cornea and most of the bulbar conjunctiva was constantly exposed. Its entire surface was dry and opaque. A superficial ulcer covered the entire cornea. The conjunctiva was edematous and bleeding from several points. The eye was immobile and the entire orbital contents and lids were very firm and tense. They pulsated with each beat of the pulse. A bruit could be heard over the left brow and temple. Compression of the left carotid artery stopped the bruit, the pulsation, and also the throbbing pain.

Stereoscopic X-ray pictures of the skull were negative. Kahn and Kline tests of the blood were 4 plus. The spinal fluid was normal and was also negative for syphilis.

The condition of the eye rapidly became worse so that it was evident that it would have to be removed. Ligation of the internal carotid was followed by an immediate improvement of all symptoms. A few days later the eye was eviscerated at which time a purulent infection was found to be present throughout the entire eye. Following this operation the reaction was quite severe and pus drained from the scleral cavity for several weeks.

About three weeks after the carotid ligation, the patient complained of increasing headaches. Lumbar puncture showed the spinal fluid cloudy and under an increased pressure of 150 mm. of water. The cell count was 915 per cubic mm., 73 per cent were polymorphonuclears and 27 per cent lymphocytes. The culture was first reported positive for the streptococcus nonhemolyticus. Subsequent cultures failed to show this organism, but instead a gram negative bacillus, which proved to be Bacillus Alkaligines. Probably both were contaminations. None of the usual signs of meningitis were present and the man was not extremely ill. Sulfanilamide was given in large doses and sulfapyridine was also tried later.

The spinal fluid gradually cleared and his headache ceased. He then complained for the first time of poor vision in his right eye. Fundus examination showed a partial primary optic atrophy the exact cause of which is somewhat doubtful. The entire temporal field of vision is lost. Vision is 20/200. Probably pressure from the aneurysm on the central portion of the chiasm was responsible, but possibly it was caused by a localized meningitis involving the anterior horn of the chiasm on its medial side. The presence of a brain abscess is seriously suspected by the attending neurologists who have examined this man. The spinal fluid cell count was 50 per cu. mm. when last taken a few days ago. The patient says he feels fine and apparently is going to recover. He is still confined to the hospital where he has now been for over ten weeks.

Syphilitic involvement of the wall of the internal carotid artery, with eventual rupture into the cavernous sinus, was the probable cause of the pulsating exophthalmus in this case.

RETROGRADE DEGENERATION IN THE OPTIC NERVES AND TRACTS

An Experimental Study of Changes in the Axis Cylinders*

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THE OCCURRENCE of retrograde degeneration in the optic nerve following its section has long been a source of discussion (Birch-Hirschfeld,[1] Ramon y Cajal[2]). In a previous paper,[3] certain aspects of retrograde degeneration occurring in the optic nerve and retina following section of the optic tracts and optic nerve were reported. These studies showed that very active demyelinization occurred in the nerve fibers of the optic nerve posterior to transection, but following section of both tracts only slight damage to the myelin of the optic nerves was observed. This would indicate two possibilities: either the optic nerves are immune to degeneration following tract lesions, or demyelinization gradually diminishes distal to the lesion and therefore is minimal or absent in the optic nerves. In support of the latter contention is the observation that degeneration after a posterior lesion of the optic tract decreased in extent as the chiasm was approached.

In order to gain further information concerning the phenomenon of degeneration, additional experiments were made on cats and monkeys. The optic tracts were sectioned anteriorly in several animals by means of the Horsley-Clark[4] stereotaxic instrument, and optic nerve section was performed at the chiasma by the temporal operative approach. Various periods of time were allowed to elapse prior to histologic investigation of the degenerating tissues. Since it was particularly desirable to determine the reaction in the axis cylinders, Bodian's[5] silver stain was used most extensively. In some instances Marchi or Held stains were used to confirm the presence and extent of degenerative changes in the myelin.

* This work is a portion of research studies in neuro-ophthalmology being made in the Department of Ophthalmology of the University of Iowa under a grant from the American Academy of Ophthalmology and Otolaryngology.

ELECTROLYTIC SECTION OF THE OPTIC TRACTS

One or both optic tracts were sectioned with the stereotaxic instrument according to the technique previously reported. Four cats and six monkeys were used in the experiments. Tissue was prepared from the animals after periods of from five to sixty days. Silver and Marchi or Held stains were made of tissue obtained from the optic nerves and tracts.

SECTION OF OPTIC TRACTS ANTERIORLY

Successful anterior section of the optic tracts (Table I) was made in three monkeys and one cat. In C645 both tracts showed extensive degeneration ten days after section, but the axis cylinders in each optic nerve were entirely normal. The lesion in this animal was made within 2 mm. of the optic chiasm, and the tracts showed great degeneration above the lesion. Marchi studies of the nerves revealed disintegration of the myelin that was much greater in the posterior portion than in the anterior (Figures 1 and 2). In the monkey M612, five days after section of the tracts, there was likewise no degeneration in the axis cylinders of the nerves. After twelve days there was no further change, and the axis cylinder remained normal. After sixty days, M629 showed some decrease in the number of axis cylinders in the optic nerve, but the usual degenerative phenomena were absent (dissolution of the entire axis cylinder and myelin sheath structures). Held stain confirmed this reduction in the number of nerve fibers present.

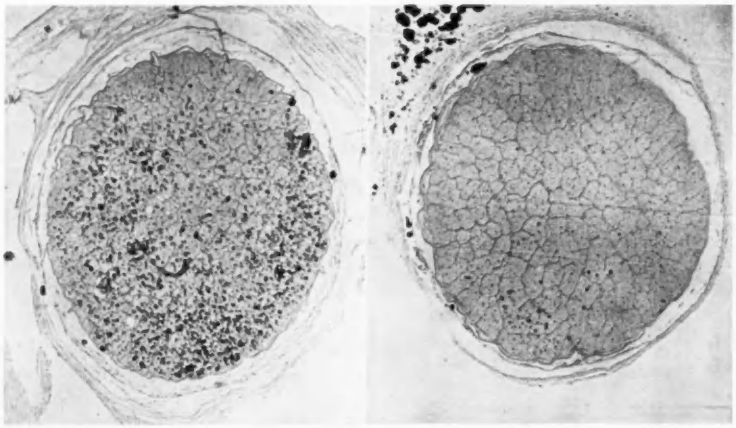

FIG. 1 — (Leinfelder) — Posterior and anterior degeneration in the optic nerve following anterior section of both tracts. (Marchi.)

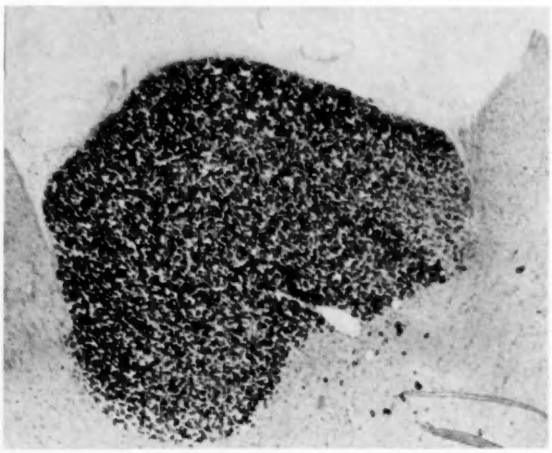

FIG. 2 — (Leinfelder) — Degeneration in the optic tract (Marchi.)

SECTION OF OPTIC TRACTS POSTERIORLY

Posterior lesions in the tracts (Table II) were accomplished in three cats and three monkeys. Sections for study were made at intervals between twenty and thirty-two days after operation.

In cat C632, successful lesions in both tracts resulted in practically complete degeneration in the posterior tracts, somewhat less in the anterior tracts, but no demonstrable degeneration of the axis cylinders in the optic nerves. Only slight degeneration was observed in the optic nerves with osmic acid stain, yet great changes were present in the myelin sheaths of the optic tracts.

In the tissue from other animals, a similarity in reaction was observed. Extensive degeneration was present in the posterior tract, a considerably less amount occurred in the anterior tract, but only a decrease in the number of axis cylinders could be observed in the optic nerves. Although the decrease in the number of nerve fibers was generalized, the thinning was more extensive in certain areas or sectors. Evidence of degeneration was less apparent in the anterior nerves than the posterior portion.

Osmic acid preparations showed only slight degeneration in the optic nerves but extensive change in the optic tracts. It was likewise noted that in the osmic acid preparations there was less evidence of reaction in the anterior portion of the nerve than in the posterior. It is definitely apparent that in all of these animals the amount of degeneration in the optic nerves whether shown by

silver, osmic acid, or the Held stain, is of little significance when compared with extensive changes in the optic tracts.

SECTION OF THE OPTIC NERVE ANTERIORLY

In six animals, four cats and two monkeys (Table III), the optic nerve was sectioned in the orbit, and in one cat the retina was removed from one eye. These experiments were performed in order to show the primary degenerative changes in the optic nerve and to demonstrate, if possible, efferent fibers that might be passing from the brain through the optic nerve.

The optic nerves of all animals showed complete degeneration of the axis cylinders (Figure 3) throughout the extent of the nerve. In the tracts the degeneration was partial because of the in-

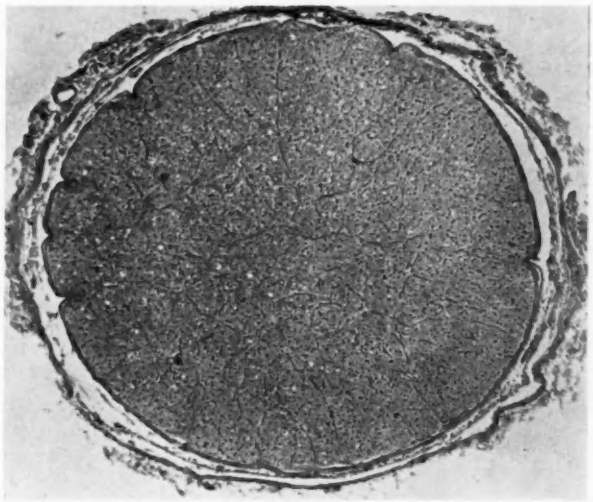

FIG. 3 — (Leinfelder) — Complete degeneration of the optic nerve following its anterior section. (Silver.)

complete crossing that occurs in the chiasm. No evidence of efferent fibers could be obtained with the silver stain. Likewise as would be expected, there was no degeneration in the opposite uninjured nerve.

SECTION OF THE OPTIC NERVE POSTERIORLY

In seven animals, one monkey and six cats (Table IV), the optic nerve was sectioned immediately anterior to the chiasm. After periods of time (ten to seventy-five days), preparations

were made of the optic nerves and tracts. Staining was with silver, osmic acid and Held. In monkey M1 the only evidence of degeneration in the optic nerves ten days after operation was an apparent swelling of the axis cylinders in the posterior sections of the nerve. The optic tracts showed definite evidence of disintegration of the axis cylinders. In cat C611 silver staining disclosed no evidence of change in the axis cylinders of the optic nerves, while extensive degeneration had occurrd in the optic tracts (Figures 4 and 5). However, after time intervals of thirty and seventy-five days following operation, there was rather extensive degeneration of the axis cylinders in the posterior portion of the nerve, but less marked involvement of the anterior portion (Fig 6).

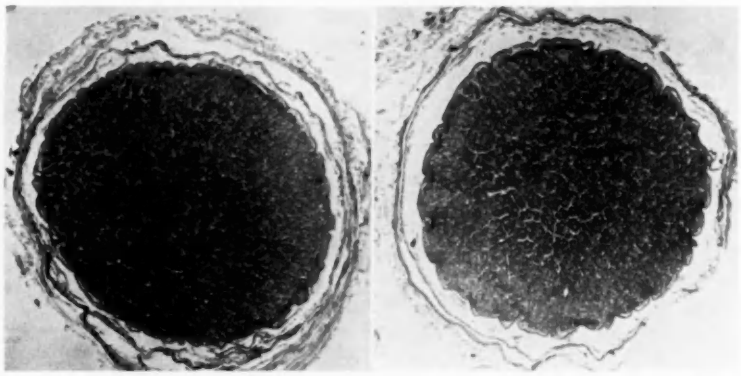

FIG. 4 — (Leinfelder) — Normal appearing optic nerves fourteen days after posterior section. (Silver.)

DISCUSSION

The data from all experiments except section of the optic nerve anteriorly, show much greater degeneration in the optic tracts than is present in the optic nerves. This at first would seem to indicate that the degree of degeneration is in some part at least dependent upon a difference in structure between the tracts and the nerves. Yet it is apparent that there is a greater degree of degeneration in the optic nerve following its posterior section than occurs in it after anterior or posterior tract section. Likewise posterior section of the optic tract results in less intense degeneration in the chiasmic region of the tract than is present at higher levels. It is possible that the greater degree of retrograde degeneration in the nerve following posterior section is in part at least due to surgical interference with its vascular supply.

FIG. 5 — (Leinfelder) — Typical appearance of degeneration in the optic tract. (Silver.)

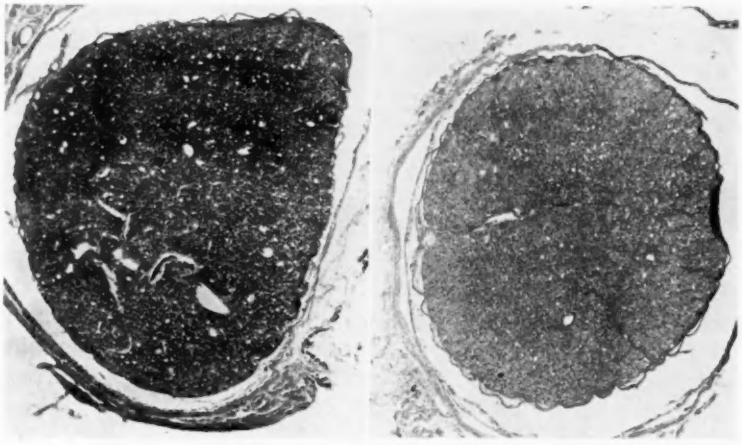

FIG. 6 — (Leinfelder) — Posterior and anterior section of the optic nerve seventy-five days after its posterior section. (Silver.)

That the retrograde degenerative phenomena are different from the primary type is well shown in Tables III and IV. Section of the optic nerve anteriorly is rapidly followed by complete disintegration of both myelin sheaths and axis cylinders while a posterior section causes no observable early change in the axis cylinder, and later results in only incomplete degeneration. It is peculiar that posterior section of the optic nerve eventually results

in a retrograde degeneration that in part bears a resemblance to primary degeneration — increased homogeneity. This phenomenon does not occur after tract lesions.

It appears, therefore, that retrograde degeneration is of an incomplete nature and that, as recognized by Boeke[6] for nerve fibers in general, it usually extends only a few segments from the lesion but occasionally a fiber may disintegrate completely. The present experiments seem to indicate that visual fibers react in the same manner. The impression is gained that the extent of retrograde degeneration in the optic nerve is a phenomenon that is dependent upon the distance of the lesion from the nerve cell, just as it is with nerves in general. Thus a lesion near the lateral geniculate body in the tract results in little retrograde degeneration in the optic nerve fibers, while a lesion in the optic nerve results in far greater destruction of the axis cylinders.

The degeneration of some of the nerve fibers, even after distant injury, is interesting. It is known that any injury to the axon results in a chromatolytic change in the ganglion cell (Nissl[7]). The more severe the injury or the closer to the nerve cell that it occurs, the more severe will be the reaction of chromatolysis (Bielchowsky[8]). Birch-Hirschfeld pointed out that many retinal ganglion cells were only temporarily injured by the chromatolytic process, and that except for an increase in the Nissl substance, they eventually returned to normal. Many cells, however, completely disintegrate. The reduction in number of ganglion cells was confirmed by James[9] and by a previous paper of the author. Although a report of the study of the retinaes is not included in this paper, the ganglion cells were observed in most specimens, and the reduction in number was never obvious after section of the optic tracts, but was definite after posterior section of the nerve. James observed a marked reduction in the number of cells after anterior section of the optic nerve. It appears therefore that the disappearance of the nerve fibers in retrograde degeneration occurs following and as a result of the death and disappearance of the ganglion cells. The incomplete nature of the degeneration along with its late development appear to substantiate this theory.

This work, therefore, indicates that the visual pathway is not peculiar in its reaction to degeneration, but that like other nerves or afferent tracts, it reacts to injury by retrograde degeneration which is in proportion to the extent of injury and to the distance of the injury from the nerve cell.

TABLE I

Degeneration Resulting from Anterior Optic Tract Lesions

		SILVER				OSMIC ACID			
		Tract		Nerve		Tract		Nerve	
	Time	Right	Left	Right	Left	Right	Left	Right	Left
M612	5	80	85	n	n	Lesion	Lesion	+	+
								++	+++
C645	10	85	85	n	Ant. n	85	80	+	+
				n	Post. n	85	80	+++	+++
M624	12	25	20	sl.	sl.	25	15	sector+	sector+
				sl.	sl.	30	25	+	+
M629	60	n	50	++	++	n	40	sl.	sl.
				++	++	n	40	sl.	sl.

TABLE II

Degeneration Resulting from Posterior Optic Tract Lesions

		SILVER				OSMIC ACID			
		Tract		Nerve		Tract		Nerve	
	Time	Right	Left	Right	Left	Right	Left	Right	Left
C616	4					90	90	sl.	sl.
C632	20	65	80	n	n	65	80	tr.	tr.
		80	95	+	+	80	95		
M628	21	n	5	n	n	n	5	n	n
		n	10	n	n	n	15	n	n
C639	23	15	90	n	n	15	85	n	n
		30	100	n	n	15	85		
M627	24	n	50	+	sl.	n	50	tr.+	tr.
		n	90	+thinned	sl.	n	100		
M625	32	n	40	+sector	sl.	n	50	very slight	
		n	50	++thinning	sl.	n	60		

TABLE III

Degeneration Resulting from Anterior Optic Nerve Sections

SILVER STAIN

		Tract			
	Time	Right	Left	Right	Left
C608	5	++ anterior	+	n	100
		++ posterior	+	n	100
MC621	10	++	+	n	100
		++	+	n	100
C626	18	++	+	n	100
		++	+	n	100
M633	20	++	+	n	100
		++	+	n	100
C634	21	++	+	n	100
		++	+	n	100
M500	87	++	+	n	100
		++	+	n	100

TABLE IV

Degeneration Resulting from Posterior Optic Nerve Sections

SILVER STAIN

	Time	Tract Right	Tract Left	Right	Left
M1	10	++	+	n	swollen
		++	+	n	swollen
C611	14	++	+	n	n
		++	+		
C630	14	++	+	n	n
		++	+	n	30%
C613	15	+	++	30	n
		+	++		
C691	20	++	+	20	n
		++	+	40	n
C603	30	+	++	20	n
		+	++	50	n
C669	75	++	+	sl.	50%
		++	+	+	80

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GLAUCOMA FOLLOWING RADIATION

Pathological Report*

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CLINICAL reports of glaucoma following the use of x-ray and radium therapy are infrequent. In a survey of the ophthalmic literature, only three pathological reports were found.

In 1921 Birch-Hirschfeld reported the case of a 61 year old man, who 20 years before had a wart on his right upper lid. This was removed in 1914, but reappeared in 1919 and grew larger. He had five 45-minute exposures to the lid in the course of one year, without protection to the globe. Two weeks after the first dose he had headache, pain in the eye, and redness of the lid skin and conjunctiva. This disappeared between exposures, but reappeared with each treatment.

When examined in May, 1920, there was an epithelialized scar on the lid whose margin was free from cilia. The lid conjunctiva was thickened, chemotic, and had numerous dilated, tortuous blood vessels. A thin, vascularized pannus lay on the upper cornea near the limbus. The cornea was opaque from deep and superficial infiltrates. Its surface was dull and smooth. The iris was atrophic, the pupil of maximum width, and the tension with the Schiotz tonometer was 40 mm. of mercury. The eye was blind. The left eye was normal.

Histological examination revealed perivascular hemorrhages from serous transudates in the conjunctiva. The basal cells of the corneal epithelium were flat and had rod-shaped nuclei. The next layer was poorly stained, the cells vacuolated, irregularly shaped, and degenerated. The external layer consisted of a row of flat cells. In some places the epithelium existed only as a single layer of cells. The conjunctiva immediately around the cornea contained

* From the Division of Ophthalmology, Department of Surgery, University of Chicago, E. V. L. Brown, M.D., Director.

plasma cells in the subepithelial tissue. There were tortuous blood vessels, some with narrowed, others with wide, lumina. The pannus like tissue extended even into the deep layers of the cornea. There was dust-like pigment on the anterior surface of the iris. The iris and ciliary bodies were atrophic and their blood vessels obliterated. The walls of the retinal blood vessels were thin, their endothelium elevated and adventitia thickened. Perivascular spaces were widened; perivascular glial fibers were separated and torn. The disc was pathologically cupped and the nerve fibers atrophic. There were fine, striated hemorrhages in the retina.

Peters reported a case of conjunctival carcinoma treated with x-ray in which glaucoma appeared one year after the last treatment. The eye was removed and studied. It revealed sclerosis of the ciliary processes which appeared short and shrunken. The fibrils of connective tissue were interspersed with thick cells which had little collagenous tissue and fine nuclei. There were some scarred, chalk-like dots. There was no sign of inflammation. The blood vessels were not diseased.

In 1904 Birch-Hirschfeld exposed rabbits' eyes to therapeutic doses of x-ray, and made a pathological study. He found partial degeneration of the retinal ganglion cells with vacuolization of the protoplasm dispersion of the chromatin substance, nucleus and cell shrinking. There was degeneration of the nerve fibers and their medullated sheaths.

He reported the case of a 59 year old male, who had a malignancy of the skin of his temple, who had six 20-minute and one 45-minute exposures to x-ray during 10 months. There was no glaucoma. The tumor extended onto the globe and the eye was enucleated. The ciliary epithelial cells were poor in protoplasm and in pigment. The iris blood vessel walls were thick and the lumen narrow. Its stroma pigment was very sparse. There was dust-like pigment on the anterior surface of the iris. The posterior iris pigment was vacuolated, swollen, and the dilator muscle very easily seen. There was no pigment in the Spaces of Fontana. A thin membrane extended from the Descemet's membrane over the pectinate ligament in a portion of the globe. The retinal blood vessels, even the central retinal vessel, had narrowed lumina. There were normal nerve fibers in the optic nerve, with a physiological excavation. The ganglion cells of the retina were involved; scarcely any were normal. There was loss of chromatin, shrinking of the nuclei, and dissolution.

This last case is mentioned to show that the pigment disturbance followed x-ray therapy to the eye without causing glaucoma. Both of these cases, as well as the experimental work on rabbits, showed similar changes which Birch-Hirschfeld believed were due to the result of blood vessel injury from the x-rays.

It was my intention to present 3 cases with changes in the eye following radiation, all of which showed parallel alterations. Time will not permit that all be given in detail.

One case, a male, 60 years old, had a sarcoma of the choroid of the right eye. The left had an incipient cataract with normal tension. He had received 600 r exposure to x-ray. The disk had a glaucomatous excavation. There was rarefaction and dispersion of pigment in the ciliary processes and iris. There was sclerosis of the pectinate ligament and pigment in the Spaces of Fontana in ⅓ of the sections.

Because this was a pigmented tumor and because any intraocular neoplasm can cause glaucoma — the case is not presented in detail.

Second case, a 46 year old woman had considerable radium Rx and no x-rays for carcinoma of the right limbus. She had a right cataract but *no* glaucoma. The left eye was normal. There was no pigment in the pectinate ligament, though the iris pigment was so markedly vacuolated that it appeared honeycombed and the ciliary pigment epithelium thinned or absent in many sections.

Report of a Case

HISTORY

A male, aged 62 years, was first seen on 5/27/36, complaining of poor vision in his left eye for six months. He thought the vision before that time was as good as the right. The eye felt irritated, but had no definite pain.

In February 1914, he had a small wart at the external angle of the right eye, which had been present for eight months and was covered by a dry crust surrounded by a mild dermatitis. This was treated by Doctor Ernest McEwen,* who gave seven x-rays or radium treatments to the lid between February and April 20, 1914. He was apparently free from symptoms until January, 1929, when there was a recurrence. Between January 17th and 26th he re-

* Doctor McEwen's (deceased) record does not state whether x-ray or radium was used, the exact dosage, or whether the eye was protected.

ceived 47 mgm. hours of radium. On February 4, 1930, there was tenderness under the scar which was stiff, and massage was recommended. On December 13, 1930, Doctor M. R. Caro found no surface evidence of recurrence. The bone at the external angle of the orbit felt roughened.

On December 27, 1930, and January 10, 1931, he received one unit x-ray (350 r) each. The eye was protected by a sheet of molded lead. On February 7, 1931, x-ray of the left maxilla showed a slight defect, which was not due to carcinoma metastasis. On June 17, 1931, Doctor C. M. Epstein excised the scar and as much tissue below and external to it as possible for histological examination.

At the operation there appeared to be a recurrence down to and along the orbital wall. Histological examination proved this to be a squamous cell carcinoma.

Between July 17, 1931, and October 20, 1931, he received a total of 300 mgm. hours of radium and 1750 r of x-ray. Several biopsies on the tissue in the first three months of 1932 showed no malignancy, but that of April 26, 1932, showed the bone to be involved. In May, 1932, three 2 mgm. radium needles were inserted, and in May 1933, three 4 mgm. needles were used, these latter to the extensions in the soft tissues of the cheek. In June, 1933, Doctor C. M. Epstein resected the left upper jaw and did a curettement of the left maxillary antrum. A plastic to repair the defect was done on May 15, 1934, by Doctors Epstein and Samuel Salinger. After the plastic there was a ½ inch opening into the left antrum, and the exposed turbinates could be seen. On September 8, 1934, May 14, June 9, August 16, September 22, 1936, and on March 3, June 14 and June 25, 1937, further diathermy resections and plastic repairs were performed by the same surgeons. On January 15, 1938, the patient developed erysipelas of the face. On January 29th he had a cerebral hemorrhage with complete left hemiplegia. On February 4th he developed lobar pneumonia, and on February 9th died. No autopsy was done.

EXAMINATION OF THE EYE

On May 27, 1936, the right vision with +3.00 sphere was 8/10-3; left vision counting fingers at 2½ feet, which no lens improved. The left lower lid was adherent to the bulbar conjunctiva just above the fornix. The cilia were inverted. There was a yellow secretion on the lid margin. The globe could neither be elevated

nor depressed, and there was limitation of both abduction and adduction. The bulbar conjunctiva in its lower one-half was beefy and chemotic. The left cornea was clear. The anterior chamber was deep. The pupil was 3½ mm. regular and reacted normally to light and accommodation. In the temporal one-third beneath the anterior lens capsule was a single vertical linear opacity. The left disc had an ampulliform excavation and a greenish pallor in the temporal two-thirds. The blood vessels bent over the scleral ring and were lost at 10 and 12 o'clock. The remaining blood vessels were all pushed to the nasal one-third of the disc. With the Schiotz tonometer the tension was 30 mm. of mercury. Aside from a small triangular superficial scar extending 2½ mm. onto the cornea between 11 and 1 o'clock, the right eye was entirely normal. On 7/13/36 there was further limitation of motion of the globe. The floor of the left orbit was partially absent. The lower lid was absent, and the skin inverted and adherent to the orbital rim. There was a marked congestion and chemosis of the lower one-half of the bulbar conjunctiva. The globe was otherwise unchanged.

On September 28, 1936, the skin of the left upper lid was edematous and the conjunctiva of the entire globe chemotic. The lid was adherent to the bulbar conjunctiva at its temporal and nasal angles. On March 1, 1937, the upper lid was more edematous, the skin injected and more adherent to the bulbar conjunctiva. All the conjunctiva of the lower lid, fornix and bulb, as far as the lower border of the internal and external recti muscles, was absent, and the inferior rectus appeared only as a short 5 mm. stump attached to the globe. On June 10, 1937, the left upper lid skin was red, very edematous and entirely adherent to the bulbar conjunctiva below the cornea, so that the cornea could no longer be seen. The tactile tension at this time was 3+. The right eye was unchanged. On June 14, 1937, the globe was enucleated. At the time of the operation, an artery at the external angle required a suture to control hemorrhage. The very friable skin at the external angle was torn, and was repaired with a silk suture. There was prompt healing of the conjunctiva.

The enucleated globe was placed in 4% formaldehyde for one hour, then transferred to Held's Solution and fixed for 24 hours.

It displaced 9 cc. of 50% alcohol, and measured 24 mm. vertically and horizontally, and 24.25 mm. anteroposteriorly. The corneal epithelium was denuded between 10 and 3 o'clock (artefact). The remainder was smooth and glistening. Involving the

inferior rectus and inferior oblique muscles was a firm cartilaginous mass, firmly attached to the sclera. This lay 8 mm. inferotemporally from the optic nerve and was 4 mm. in diameter. The tissue mass involved the inferior rectus to within 8 mm. of its insertion, and the inferior oblique to within 6 mm. The latter was very thin, taut, and fixed by the mass to the sclera. The mass was yellowish gray. The optic nerve cut on the oblique measured 10 mm. in length, and had a diameter with its sheaths of 2 mm. The remainder of the sclera was pearly white. The inferior vortex vein could not be located, and was probably covered by the tumor mass.

A large calotte measuring 23.75 mm. anteroposteriorly and 24 mm. horizontally was removed above. The cut surface of the globe permitted a view of the anterior chamber which measured 5 mm. horizontally and was 0.5 mm. deep. The sclera measured 1 mm. in thickness. The choroid was everywhere in situ. The lens was in situ, had a golden sheen, and its zonular fibers appeared to be normal. The retina was detached, except for 1 mm. around the optic nerve, leaving a 3 to 5 mm. empty subretinal space. It was uniformly gray. The cavium oculi was only partially filled by a filmy gelatinous vitreous attached by a few strands to the retina and opticus. The partially exposed opticus had a definite glaucomatous cup. The ciliary processes appeared whitish.

The inferior calotte measured 16 mm. anteroposteriorly by 16.25 mm. horizontally, and revealed the same structures as the superior, except that the anterior chamber was not exposed and the plain part of the ciliary body had been cut across.

Eight hundred sixty-five sections were cut at between 14 and 16 mu. Every tenth section was stained with hemotoxylin and eosin. Several sections were stained with Mallory's, van Giesen's, Pal Weigert's, Verhoeff's, Masson's trichrome, and Alfiori's depigmentation methods.

ESSENTIAL CHANGES — CORNEA

The epithelium in its thickest portion was five to six layers of cells thick, and at its maximum width measured .081 mm. In a few sections it was reduced to a single layer of cells, and was absent due to artefact in parts of several sections. The basal cells were almost square, though some were columnar. There were a few clear spaces between cells. The cells themselves were poor in cytoplasm and their nuclei granular. Those cells just above the basal cell layer were narrow and flattened and contained oval nuclei. The

outer layer was very thin and flat and had elongated oval nuclei with occasional vacuoles. Bowman's membrane was granular and indistinct in its outer one-half beneath the area of greatest epithelial destruction. In most sections the substantia propria appeared normal, though in some the fixed cells were poorly stained. The endothelium was normal for the most part; in other places the cytoplasm was granular and the nuclei fragmented. In many sections there was fine, dust-like pigment on the endothelium near the chamber angle.

ANTERIOR CHAMBER

The anterior chamber was of varying depth, being deepest in the center where it measured 1.39 mm. There was slight amount of fibrinous material on the back of the cornea in a few sections, and over the iris in two. There was scarcely any free pigment in the anterior chamber.

PECTINATE LIGAMENT

Not all the fibers of the pectinate ligament were sclerosed, though some fibers seemed slightly thicker than others, the nuclear stain poorer, and many of the Spaces of Fontana narrow. In every section the Spaces of Fontana contained granular brown pigment.

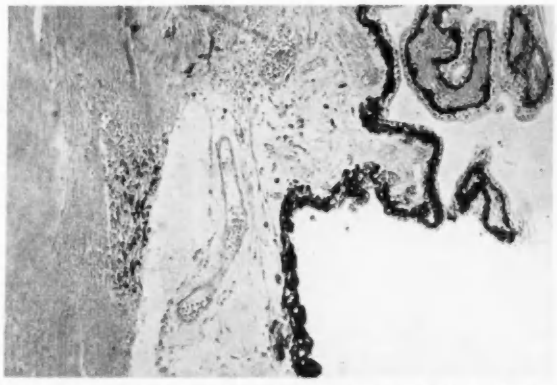

FIG. 1 — (Bothman) — Pectinate ligament with Spaces of Fontana containing pigment.

The Canal of Schlem was normal and contained no pigment. The pigment found in the chamber angle was of irregular shape and size. It varied from single grains to clumps of granules which lay on the fibers of the pectinate ligament and in the Spaces of Fon-

tana. That it was not artefact is shown by its presence in every section. It was dissolved with the Alfiori depigmentation method. The remainder of pigment at the angle was scattered like dust on the endothelium.

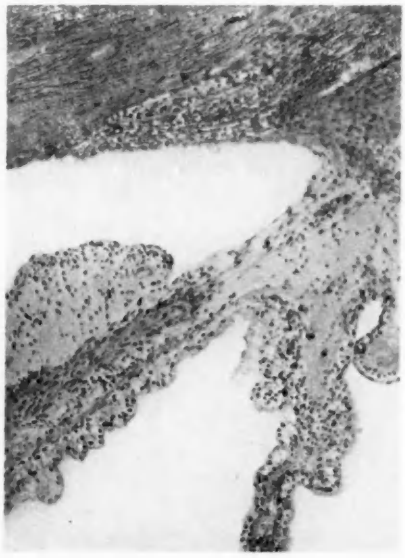

FIG. 2 — (Bothman) — Pectinate ligament with depigmentation showing Spaces of Fontana.

IRIS

The iris was very thin and rarefied throughout. Crypts appeared to be normal. There was almost no chromatophore pigment and there were only a few clump cells. The pigment which covered the anterior iris surface was fine and granular, and it was rare to find even a well-formed clump cell here or near the chamber angle. On the posterior surface the pigment was vacuolated and granular because of its dispersion. The iris blood vessels appeared to be normal. There were no anterior or posterior synechiae. Dilator and sphincter muscles were normal. In one area the pigment just behind the sphincter was almost absent, due to a disturbance of the capillary bed (Section 170). In Section 120 there was only a single layer of pigment on the posterior iris. Extending from Sections 150 to 180, there was an oval, honeycombed pigmented area

at the root of the iris connected with a posterior pigment layer. This measured 0.12 mm. anteroposteriorly by .69 mm. radially by approximately 0.33 mm. vertically.

FIG. 4 — (Bothman) — Iris showing rarefaction of the posterior pigment layer and pigment on anterior surface.

CILIARY BODY

The ciliary muscle was approximately normal in all but a few sections where there appeared to be a pressure atrophy. In most sections there was pathological depigmentation of the ciliary processes, with very poor staining and some vacuolization of the unpigmented epithelium. The lens capsule and lens epithelium were everywhere intact and there was no demonstrable cataract.

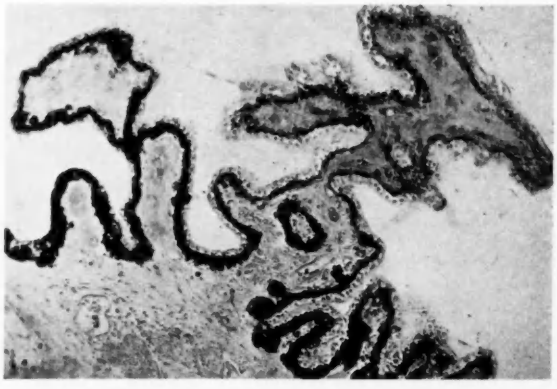

FIG. 3 — (Bothman) — Ciliary processes showing loss of pigment with changes in the non-pigmented epithelium.

CHOROID

The choroid was shrunken and thin throughout, from formaldehyde fixation, except in the area of the tumor metastasis. The choroidal blood vessels showed moderate sclerosis. The pigment epithelium was rarefied and granular. There were many proliferations of the lamina vitrea. The pigment over these was especially granular. The choroid was adherent to the retina in several places. The pigment epithelium was flattened and granular, but still present in these areas, and there were no breaks in the lamina vitrea. Opposite the external tumor the choroid contained a dark brown granular mass which measured 3 mm. horizontally by 2.97 mm. anteroposteriorly. A tumor began about 12 mm. behind the ora serrata. When depigmented by the Alfiori method, this growth was seen to be a squamous cell carcinoma by direct extension through the sclera.

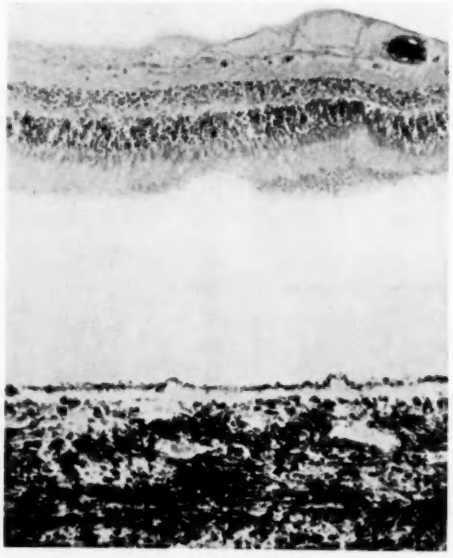

FIG. 7 — (Bothman) — Pigmented area of the choroid showing its invasion by tumor cells.

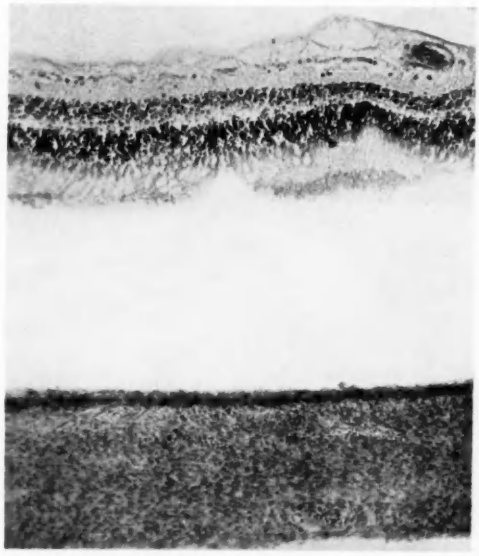

FIG. 8 — (Bothman) — Depigmented area in the choroid showing the tumor cells.

RETINA

The retina was atrophic. Nuclear layers stained normally. In the area opposite the choroidal tumor the internuclear layer was vacuolated and 2½ times as wide as the outer nuclear layer. The retina was adherent to the choroid in several localized areas. Here the rod and cone layer was granular, vacuolated, and epithelium pigment thin or absent. The ganglion cell layer was thin, granular and vacuolated. The blood vessel walls were of about normal thickness; none were twice the thickness of their lumina. There were many cysts near the ora serrata on both sides.

OPTIC NERVE

The optic nerve was pathologically cupped and its nasal side even undermined. At the scleral ring the cup measured 0.78 mm. and at the lamina cribrosa level .91 mm. The nasal side of the cup was .83 mm. and its temporal side .51 mm. deep. Its floor reached the lamina cribrosa. In Section 390 the floor of the excavation was bridged by a blood vessel from the nasal side. The Pal Weigert's

FIG. 5 — (Bothman) — Optic nerve showing the glaucomatous cup with small area of medullated nerve fibers. Periphery left side. (Pal Weigert stained.)

stain in Section 469 showed only two areas stained dark blue. One near the sclera measured 1.15 mm. by .97 mm. and another area along the edge of the nerve was .16 mm. wide by 1.64 mm. long.

TUMOR

The tumor reached its maximum size in Section 730. It measured 2.59 mm. anteroposteriorly by 10 mm. horizontally by 2.12 mm. vertically. Section 190 showed a beginning small, round, inflammatory cell infiltration of the inferior oblique muscle. Section 360 was the first section showing the tumor cells in the muscle. Section 450 showed the beginning involvement of the episcleral tissue by the growth. Section 680 showed beginning invasion of the sclera with tumor cells along the blood vessels and lymphatics, and in Section 710 the sclera was definitely thickened by the invading mass. There was hemorrhage between the tumor and the sclera. In Section 770 the sclera was invaded for .06 mm. In Section 780 there were epithelial pearl nests in the sclera. There were lymph spaces containing blood, new blood vessels, and a small, round, inflammatory cell infiltration in the invaded sclera, as well as tumor cells.

The tumor itself was made up of squamous cells with many mitotic figures invading and attached to the muscle. Scattered hyalinized epithelial cells were present, also some new blood vessels and groups of small, round cells about the vessels.

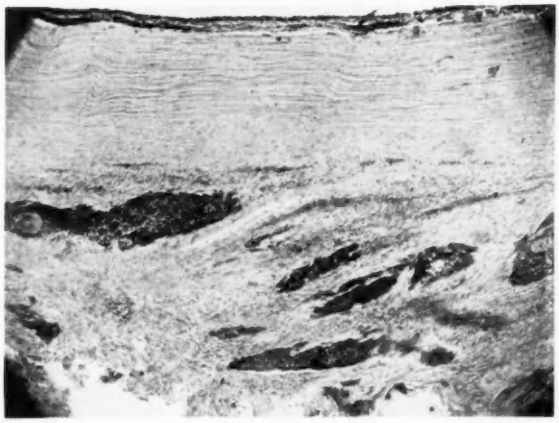

FIG. 6 — (Bothman) — Tumor invading the sclera showing epithelial pearls.

SUMMARY

A male, aged 62 years, who had large doses of x-ray and radium, much without protection, developed an unilateral glaucoma.

Histological study revealed: Thinning and destruction of corneal epithelium and some damage to the endothelium, atrophy of the ciliary processes, very marked dispersion of pigment from the ciliary processes and posterior iris, with blocking of the Spaces of Fontana and the anterior surface layer of the iris, with a resultant glaucoma. There was a complete and total glaucomatous optic atrophy, moderate choroidal vessel sclerosis, and atrophy of the ganglion cell layer of the retina. The squamous cell carcinoma invasion of the sclera and choroid was incidental and must have occurred late in the disease.

DISCUSSION

Was the glaucoma due to direct action of the rays on iris and ciliary body pigment causing its dispersion?

The fact that the blood vessels were little changed from those of a patient of the same age with normal tension would indicate that vascular changes played no role in this case.

Corneal epithelium is avascular, yet x-ray and radium produced severe damage in this and all reported cases, as well as in rabbit eyes. It would seem that alterations from radiation are not secondary to vascular changes.

In Birch-Hirschfeld's cases, both with and without glaucoma, there was a similar change in the pigment epithelium, as well as in the blood vessels of the iris and ciliary body. He believed that the pigment disturbance was secondary to the vascular change, yet in his experimental work on rabbits he described a gushing forth of iris and ciliary body pigment without vascular change. It is true that pigment disturbance is seen in chronic glaucoma of unknown etiology, but most of these have anterior synechiae. It is true some cases of glaucoma show only sclerosis and some pigment in the Spaces of Fontana with deep anterior chambers. These are not the usual findings, however, and most often the other eye also has glaucoma.

Whether the ganglion cell damage was due to radiation or a result of glaucomatous pressure, is also difficult to decide. The vacuolization of these cells, their shrinking, hyperchromatosis of their nuclei and dispersion of the chromatin substance occurred in rabbit retinae. These changes occurred in areas near blood vessels and in others free from retinal vessels, so that they were not dependent on vascular changes. The changes occurred clinically at a slower rate than those in experimental animals. The same type of change occurs clinically with quinine, methylalcohol poisoning, and some post-mortem eyes, so that they may have been due to direct action of the rays on the retina. However, like changes can be due to glaucomatous pressure atrophy.

Why radiation produces cataracts without glaucoma in some cases, glaucoma without lens changes in others, or both in some eyes, is not known. In still other eyes with normal lenses or with aphakia, retinal atrophy may follow radiation. It is possible that there is a selective action of the rays for certain structures, but such selectivity has neither been explained nor can it be predicted.

CONCLUSION

A case of glaucoma which followed radiation therapy is presented. Minor doses of x-ray were given in 1914, 1929 and 1930, and large doses in 1931, 1932 and 1933. The diagnosis of glaucoma was made on the first visit in 1936. There was no tumor in the eye when the fundus was visible. The glaucoma was unquestionably present before the choroid was invaded. The choroidal involvement consisted of a slightly less than 3 mm., almost round tumor. The right eye remained normal. The outstanding pathological finding was the breaking up of anterior uveal pigment epithelium,

and its dispersion over the anterior iris and in the Spaces of Fontana with very little vascular change and no synechiae. The three recorded cases, and my two cases briefly outlined, as well as the last, all showed similar alterations of anterior uveal pigment. It is assumed that the pigment disturbance was due to the rays, and the glaucoma in the last case was a mechanical one due to *blocking* of the aqueous escape from the eye.

We wish to thank Doctors Caro and Epstein for the use of their records, Doctor E. V. L. Brown for his help with the pathological study, and Mrs. A. Carroll for the preparation of the specimen.

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DISCUSSION

WALTER E. CAMP, M.D., Minneapolis: I thought that as long as we are considering irradiation being the cause of glaucoma, it might be well to summarize our present knowledge of how irradiation affects the tissues of the eye.

This is a summary, not only from clinical cases but also from experimental work, showing our present knowledge of the effects of irradiation on the eye and the sensitivity of the tissues. I think it is pretty well conceded that the eyes of all experimental animals are much less sensitive than human eyes.

Antenatal irradiation to mother may produce questionable congenital anomalies of the eye and congenital cataract. Irradiation of rapidly growing eyes of young animals soon after brith show: (a) Retardation of growth in size of globe. There is a marked retardation in all the tissues of the globe for a considerable time. This retardation is greater the younger the animal. (b) Marked and early cataractous change in the lens. It seems that in the young individuals the lenses are much more sensitive to irradiation than they are in older individuals.

The conjunctiva and lids are more sensitive at all ages than are the remaining structures of the eye or the surrounding skin of the face. To me it was quite enlightening to know that the lids are more sensitive than the skin around the face.

The sensitivity of the cornea is next in order. The iris is next in order of injury, which consists of myosis, disturbance of iris pigment and degeneration and swelling of the endothelium of blood vessels. The myosis is very resistant to drugs and may even be permanent. Apparently the damage to the blood vessels comes late and after very high dosage. In young

animals the retardation of pigment formation is quite constant, both in experimental animals and in clinical cases. In both experimental animals and in humans the iris is affected only by large doses of radiation.

In adults the lens is quite resistant to radiation and the retina is least sensitive. Pathologic changes frequently reported are usually due to postmortem change. The retina, like the entire central nervous system, is quite resistant.

It would appear from Doctor Bothman's slides that we are dealing here with a case in which radiation was a possible cause, although one has to be quite careful in ruling out any other possible cause for the condition.

From a study of the material available, it is apparent that when the uveal tract is damaged by irradiation, it is the uveal pigment that is chiefly affected. In the fetus and infant pigment formation is greatly retarded. It is probable, as Doctor Bothman emphasizes, that it is this pigment dispersion rather than sclerosis of the meshwork of the iris angle that accounts for the chronic glaucoma. In view, however, as in this case, of the invasion of the orbit and globe by a neoplasm one should hesitate to attribute the glaucoma to irradiation alone.

GEORGIANA DVORAK-THEOBALD, M.D., Oak Park, Ill.: Permit me to present several slides with a normal trabeculum, showing, (1) openings from the anterior chamber into the inter-trabecular spaces; (2) the inner canals of Sonderman connecting the inter-trabecular spaces with the Canal of Schlemm; (3) the external collector channels which connect the canal with the anterior ciliary veins. Aqueous humor reaches the canal of Schlemm by way of the inter-trabecular spaces and the inner canals. When the inter-trabecular spaces become occluded, increased intraocular tension results. The occlusion may be due to a swelling and sclerosis of the trabeculae, or to a blockage of the intertrabecular spaces by foreign substances.

In the specimen presented by Doctor Bothman, the inter-trabecular spaces are completely blocked with degenerated pigment, derived from the pigment epithelium of the iris and ciliary body which are atrophic. The trabecular fibres are sclerosed, hazy and stain poorly.

Disturbance caused by irradiation may be due:

(1) To hyperemia and subsequent inflammation
(2) To obliteration of the blood supply and atrophy of the adjacent tissues; and
(3) To direct action on the nuclei of the pigment epithelial cells themselves.

When the nutrition of uveal pigment epithelium is interfered with, these cells may proliferate, disintegrate, and migrate.

The pathology of this eye is such as is found in the late stage of severe glaucoma, secondary to pigmentary degeneration without regard to etiology.

Section on Otolaryngology

The American Academy
of Ophthalmology and Otolaryngology

Forty-Fourth Annual Meeting
Chicago · 1939

RESEARCH REPORT ON THE ANATOMY AND PHYSIOLOGY OF THE TONGUE

MAX A. GOLDSTEIN, M.D.
ST. LOUIS

IN THE entire realm of human physiology there is no muscular mechanism of greater complication, more varied functions, unusual motility and importance in social contacts than the tongue: nor is there one organ that has been less studied and understood and more generally discussed in all epochs of science. Its intimate association with the mechanism of speech, its service in salivation, mastication, deglutition and respiration justifies all thought, energies and research that can be applied towards its better understanding.

The actual production of oral speech has been described by the earliest physiologists and linguistic authorities as the expression of vowel and consonant elements, syllables and sentences, activated by mental processes; and the tongue has always been regarded as the most vital instrument for this act. In fact, the origin of speech has been so intimately associated with the functions of the tongue that the Latin name *lingua* constitutes its etymological derivation. Thus we have *linguistics, language, zungen, tongues* — to *express speech* of the languages of the peoples of the world.

Yet, with all this valuable evidence of the speech functions of the tongue, comparative anatomy of this organ offers ample data that, throughout its evolutionary development, a complicated structure is found endowed with flexible, extrinsic and intrinsic muscles, attached to a rudimentary hyoid bone, innervated by the hypoglossal nerve, capable of serving the animal in vital processes other than speech.

In a carefully prepared report of the literature on the comparative anatomy of the tongue, Jacqueline Keaster* has reviewed and presented many details and differentiations in the evolution of

* Dissertation presented by Jacqueline Keaster of the Faculty of Central Institute for the Deaf to the Graduate School of Washington University for her master's degree.

tongues of animals of various genera from amphibians through mammals to man and has embodied the observations of Merkel, Sonntag, and MacGregor, in a dissertation prepared under my personal observation. I quote liberally from this well-arranged research work, made possible by a grant from this Academy.

Negus, in his treatise *The Mechanism of the Larynx*, traces the evolution of the larynx to its present status as an important mechanism of voice and points out that it originally functioned as a protecting valve to the deeper respiratory tract. He states: "The larynx is generally known as the organ of voice and yet it takes but a moment's reflection to observe that the thousands of species which have a larynx never — or practically never — make use of voice."

In such observations involving two necessary factors in voice and speech, the tongue and the larynx seem to have undergone evolutionary changes from entirely different primitive functions to that of apparently indispensable mechanisms of human speech.

To illustrate these evolutionary changes in the tongue and the explanation of such changes, I have selected a small number of animal tongues to emphasize some of their outstanding features.

These processes may be best illustrated by moving pictures of the tongues of animals in which feeding habits, environmental conditions and structural peculiarities are evident. Here are significant indications that the complicated tongue mechanism must serve other purposes in the animal economy than speech.

In the film that is now presented the tongue of each animal and its special function is emphasized.

FILM — ANIMAL TONGUES

Chameleon

The tongue of the chameleon has both intrinsic and extrinsic muscles; it can be thrown out of the mouth with rapidity and with a hinge-like motion, bringing food and flying insects farther into the food channel; protrusion is accomplished by the action of the genio-glossi and retraction by the action of the hypo-glossi.

Ant-Eater

The tongue of the ant-eater is characterized by extrinsic muscles with a high degree of specialization and a motility greater than that of all other mammalian tongues. Its unusual protrusion enables the animal to collect large quantities of its favorite insect food with a great extension and wide sweeping arc of its tongue.

FIG. 1 — (Goldstein)

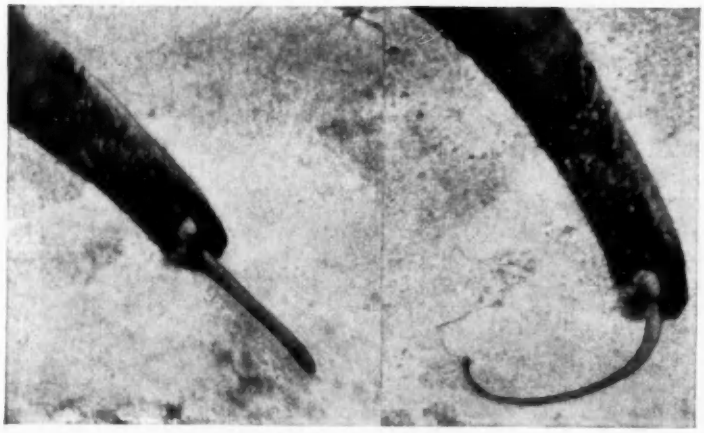


FIG. 2 — (Goldstein)

FIG. 3 — (Goldstein)

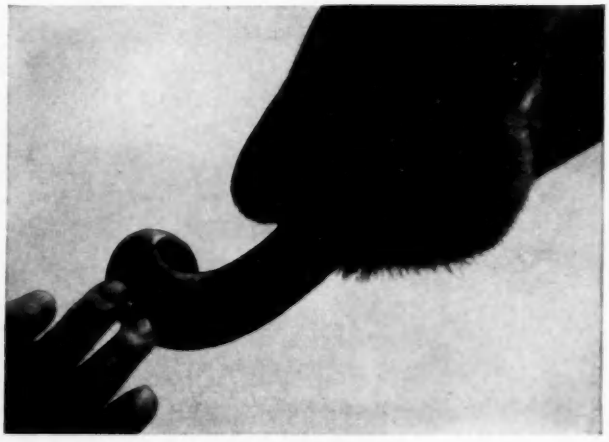


FIG. 3B — (Goldstein)

Giraffe

The structure of the giraffe, his unusual height, emphasized by long legs and longer neck, enables him to reach upwards, grasp the leaves and small shoots of tall trees with his tongue, wrap this very long, elastic compound muscle around his food and convey it to his mouth.

FIG. 4 — (Goldstein)

Elephant

When the tongue is protruded in most mammals the apex is seen. The elephant's tongue is an exception; here the apex is bound down to the floor of the mouth by a short, heavy frenum. The elongated trunk overcomes this handicap by depositing food and drink further back in the cavity of the mouth.

Parrot

Parrots have the largest tongues among birds, due to fat, vascular supply and glands, rather than to any special muscular development. The tongue mass is bulky and should, therefore, be unwieldy; the surface is rasp-like, enabling the animal to pull insects and grubs from rotten wood. Equipped with these peculiar structural features, it is unusual to note the extreme facility of the parrot in the production of articulate speech.

Mammals differ from the lower vertebrates in mastication, in salivation and in their licking power. The construction of the mammalian tongue, therefore, differs greatly in shape and size to facilitate these higher developed functions.

The tongue may be conical, spatulate, triangular or vermiform. The apex may be round, flat or pointed; entire or not. It may be small, narrow and deep, or wide and shallow. Papillae are present in all mammalian tongues and are of three varieties: Circumvallate, fungiform and conical. The functions of the papillae are

many; taste is not one of them. Papillae are tactile, retentive or mechanical; the functions differing in different animals.

Briefly correlating the differences in the structures of animal tongues we note that such differences conform to the habitat and environment of the animal.

In the frog, lizard, snake and ant-eater, the protrusion of the tongue is made possible by over-development of the genio-glossi muscles.

In the camel and giraffe the mobility of the anterior third of the tongue, slender and of special muscular strength, enables these animals with elongated necks, feeding on the leaves of tall plants, to wrap the tongue around such food and convey it to the mouth cavity. The canine tongue is abundantly supplied with mucous and salivary glands, bathing its epithelial surface and aiding absorption in this area.

It has often been observed that "the dog breathes partly through his tongue." The elephant substitutes his trunk for the blunt, stubby tongue apex and short frenum, in carrying food to the mouth.

The tongue of the whale is the simplest in construction of all mammal tongues. It is the only mammal that does not masticate; therefore, the whale tongue is not developed beyond the lower vertebrate level.

In the mammalia, the functions of mastication, insalivation and licking are dominant, and the structure of the tongue has been developed to conserve these functions.

In man, the development of articulate speech has necessitated evolutionary changes in tongue musculature, shape in all parts of the oral cavity, development of resonators and laryngeal details. It is quite evident from these numerous changes that the complicated processes of articulate speech are not dependent on the functions of the tongue alone.

We have thought and taught empirically for over a century that the tongue is absolutely essential in the production of various consonant elements. For example, to produce the consonant *t* or *d* the tip of the tongue must touch the upper incisor teeth; for *k* and *g* the middle third of the tongue is arched upward to approximate the curved shape of the hard palate; for *L* the nose is blocked off, the tip of the tongue is placed behind the upper incisor teeth and pressed against the palate, the tongue thus dividing the voice out-

put into two parts. All other consonant elements have been given their empirical allotment in tongue positions.

The question now under discussion is: "Can these consonant elements be produced without the aid of the tongue and how is this mechanism made possible?"

In 1791, Wolfgang von Kempelen, of Vienna, published a remarkable dissertation entitled "Mechanism of Human Speech and a Description of His Speech Machine." Introductory chapters in this volume include the Origin of Speech; The Various Human Instruments with which Speech is Produced; An Analysis of the Vowel and Consonant Elements, their Structure and Production, and, finally, a Description of his Speech Machine and the Various Mechanical Devices which he used as substitutes in the Production of Articulate Speech.

The ingenious and practical devices and gadgets used in the construction and performance of mechanical speech are of such up-to-date value and instruction that I have taken the liberty of illustrating and describing some of the more important features of this apparatus.

LANTERN SLIDE (PLATE) 1

In the first lantern slide here shown, we see a diagrammatic illustration of the several vowel elements (German), as formed in the larynx, projected into the mouth cavity and modified by the size of the openings effected by the lips, teeth and lower jaw, thus changing the shape and size of the mouth cavity, acting in the capacity of a resonating cavity.

Note the graduated measurements that indicate the size of the mouth opening from *U* (o͞o) minimum to *A* (å) maximum.

LANTERN SLIDE (PLATE) 2

In the woodwind instruments (oboe, flute and clarinet), the mouth represents the larynx; the reed mouthpiece, the glottis. The mouthpiece functions in the mouth, and the lips are the bands and muscles that regulate the tone and simulate the functions of the larynx. Fig. 2, *b* and *c*, represent the change in the size of the opening in the keys of the instrument to regulate the egress of air in forming the consonant *F*. This is further diagrammatically illustrated in Figs. 3 and 4, the point of egress indicating the position of the upper and lower teeth.

Fig. 1

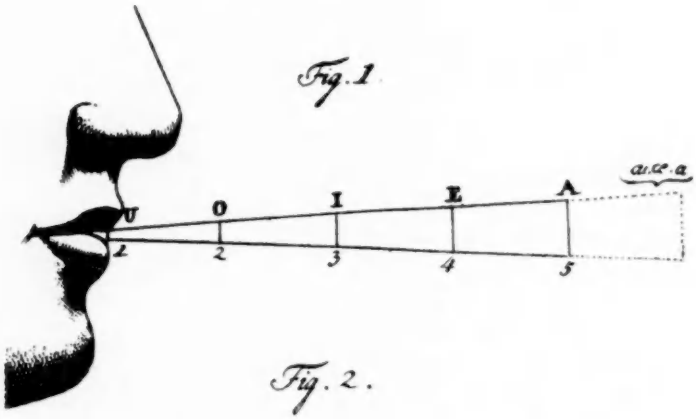


Fig. 2.

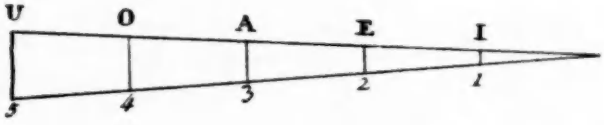


Fig. 2.

Fig. 3.

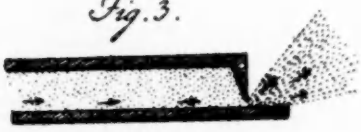


Fig. 4.

LANTERN SLIDE (PLATE) 3

We have here a diagrammatic representation of the position of the tongue in the production of the speech elements *D* and *T*. The anterior third of the tongue shown in the black, shaded portion is in apposition to the front of the plate, and by a puff of breath,

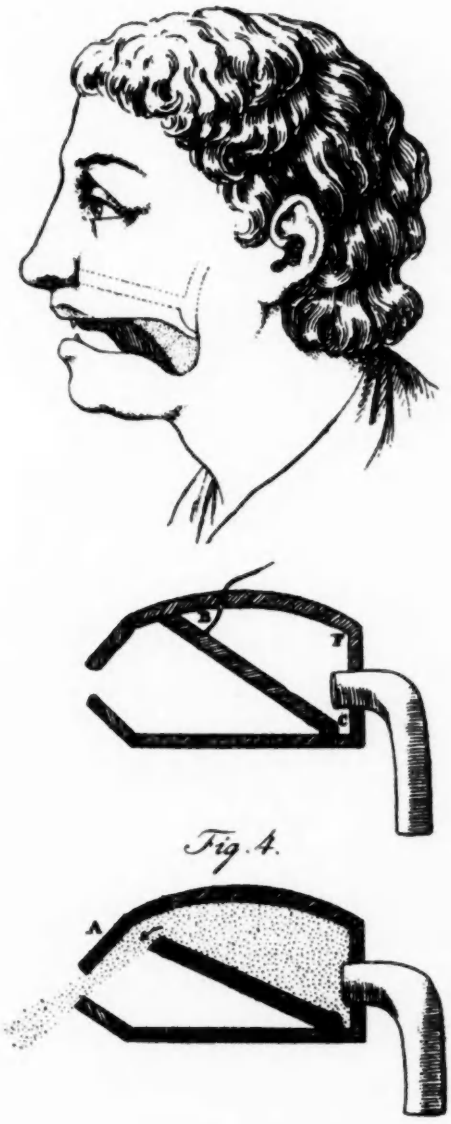


Fig. 4.

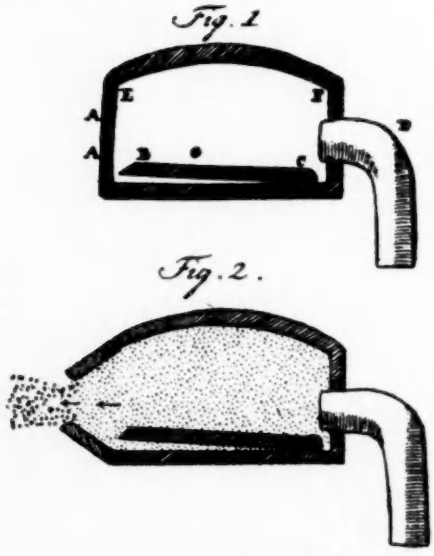
the air (dotted area) is forced between the surface of the tongue and the palate, regulated by the tongue channel.

In *D* the mouth and nose are open. *T* is an explosive *D*.

LANTERN SLIDE (PLATE) 4

This is an ingenious arrangement of the mouth cavity as represented by a wooden box shown in the shape indicated in the diagram. At *C* there is fastened a hinge that operates a thin, flat, oblong slip of wood simulating the human tongue at *b* and *c*, which closes or opens the space between the tongue and palate and in Fig. 4 in slide 3, allows the air exit in the production of the consonant *B*. A curved tube in the back of the mouth at *e* admits the air and at *a* (lips and teeth), permits its escape from the mouth.

In *B* the lips and nose are closed. *P* is an explosive *B* entirely differentiated by a voiceless puff of air. In *B* and *P* the breath is forced through closed lips.



LANTERN SLIDE (PLATE) 5

The upper illustration in this slide shows the tongue with a median groove for the escape of air from the mouth at either side of the median line.

In the lower gadget to produce the consonant *L* the thumb is placed in the trapezoid box to serve the same mechanics in spreading the air current in two directions.

The vocal cords vibrate; the nose is blocked; the anterior third of the tongue presses against the palate; the posterior mass rests irregularly on the floor of the mouth thereby changing the direction of the sound. When the flat apex of the tongue with its tip pressed against the palate and the main tongue mass rests irregularly on the floor of the mouth, a vent is created around the posterior molar teeth through which the breath, either sibilant or vocalized, passes out and thus produces the consonant *L*.

LANTERN SLIDE (PLATE) 6

The consonants *M*, *N* and *NG* are produced by varying the positions of the tongue to permit the escape of a vocalized column of air into the nasal cavities. *M* in its production is the simplest and easiest of the consonant elements.

In the speech training of the deaf child it is the first element taught because of its simplicity of production and its comprehension by the child's sense of touch, through bone conduction. The vocal cords vibrate; the nose is open; the tongue is in a position of rest; the teeth slightly apart; the lips closed.

In *M* the nose is the main phonetic factor, for, if the nose is occluded a perfect *M* is impossible. The tongue plays a minor role in its production. Perfect phonation of *M* depends on vocalized breath, unobstructed naso-pharynx, closed lips.

As in *M* the consonant *N* requires an unobstructed nose and normally functioning vellum palati. In *N* the vocal cords vibrate; the nose is open; the tongue channel is closed by pressure of the tip and anterior third against the palate; the mouth is open.

In *M* the closed lips block the tongue channel; in *N* the tongue performs this function. Both require a patulous nose.

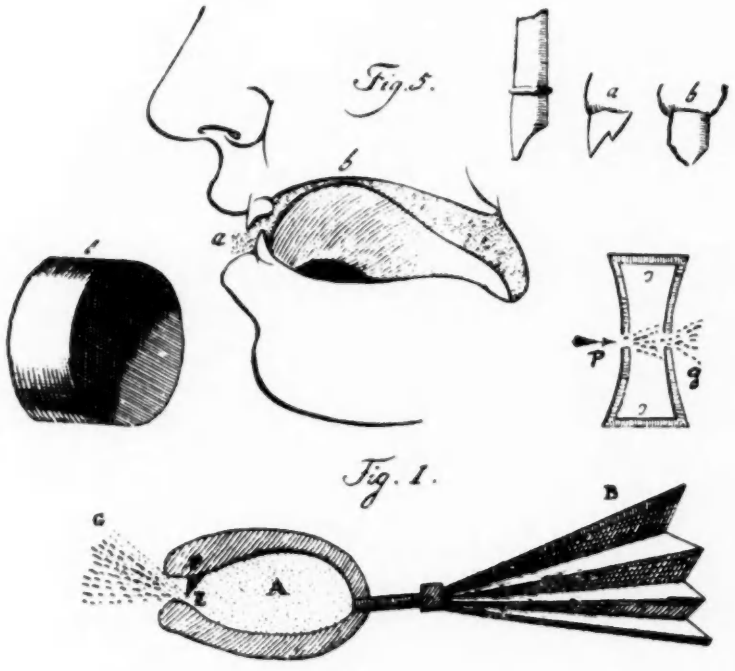
LANTERN SLIDE (PLATE) 7

To produce the consonant *S* the diagram on the right side of the slide shows the ingress of air at *g* and the egress of air at *f*. Note the difference in the size of the openings and the compression of air as it passes through the opening between the teeth at *f*. In the upper right hand corner of the slide *a* and *b* represent the incisor teeth. In the human tongue the slightly arched tip and anterior third control this air egress (as seen in the central diagram) and carries out the principle illustrated in the diagrammatic sketch to the right.

The lower illustration shows the bellow attached to the mouth cavity and the escape of air from the lips and partially closed teeth at *e*.

In *S* the anterior third of the tongue is pressed against the palate; the tip directed downward toward the lower incisor teeth; the breath is not vocalized; nose is closed; lips apart; teeth not absolutely necessary but help to accentuate this consonant.

The consonant *Z* combines the consonant *S* with a final vowel (e) or in the middle syllable of a word with an initial vowel. In short, *Z* combines the sound of *S* with a vowel, either initial or final.

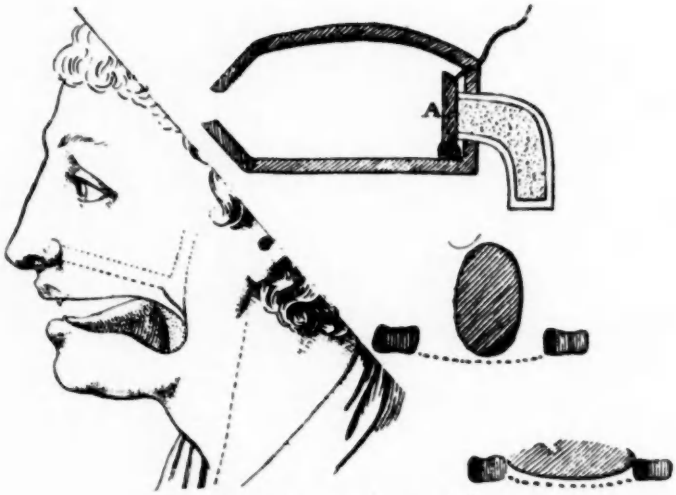


LANTERN SLIDE (PLATE) 8

This slide illustrates the mechanical production of *K*, the most difficult of the consonant elements, especially in the training of the deaf child.

In *K* the lateral margins of the tongue are elevated; the middle third is arched upward toward the palate; the tip touches the lower incisor teeth; the posterior third touches the soft palate and blocks the passing of air to the pharynx. The sudden puff of the column of air from the larynx aided by pressure from the lungs and diaphragm, and the sudden release of the tongue from the soft palate, produces *K*.

To convert *K* into *G* (hard as in *go*), requires only the vocalization of breath as described in the mechanics above.

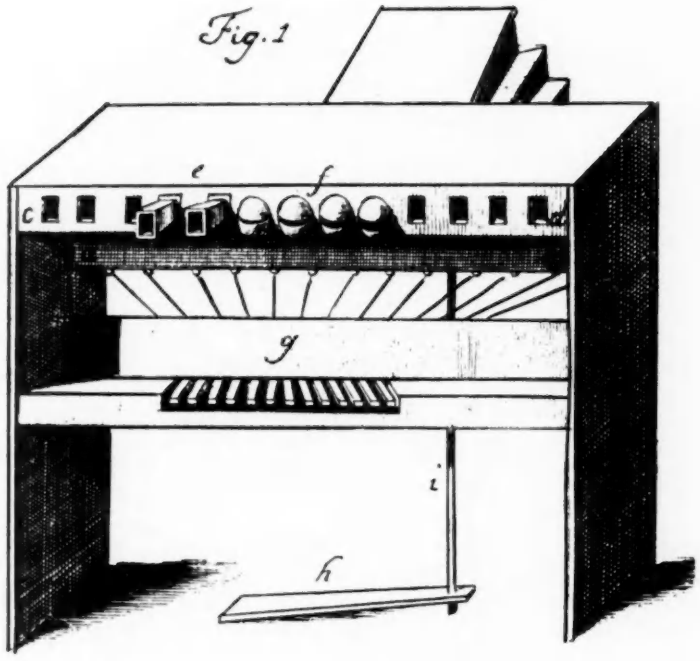


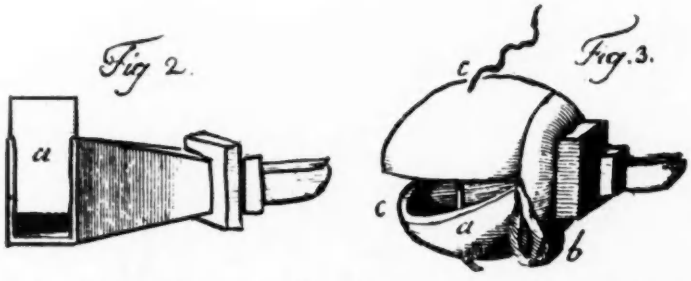
LANTERN SLIDE (PLATE) 9

In Fig. 1 of Slide 9 is illustrated the Kempelen Speech Machine, ready for operation.

From *c* to *d* are thirteen oblong-shaped openings connected with a broad resonating chamber; *e* shows two of the individual wooden gadgets that fit air-tight into each of these openings, illustrated in more detail in Fig. 2. At *f* are four accessories, composed of two halves of a rubber ball (Fig. 3, *a* and *c*), which are hinged and fitted tightly to a connecting piece, each of which fits into individual holes in the sounding board. A cord is passed from *a* to *c* and regulates the opening and closing of this rubber ball to simulate the individual vowel elements. Each vowel is supplied with its own gadget of which there are four. At *g* is the keyboard of thirteen keys, which operates the thirteen openings in the resonanting chamber. The bellows is shown in the rear and the foot pedal at *h*. The air control is produced by the foot pedal operating the bellows. The release of the cords operates the individual gadgets in the resonating chamber and is controlled by the hands on the keyboard at *g*.

In the light of modern engineering skill and electric and acoustic devices, it is a remarkable observation that the Kempelen Speech Machine, with its simple mechanics and in the hands of an experienced operator, could produce results that closely simulated human speech, nearly 150 years before the appearance, a few months ago, of the VODER, a modern speech machine, activated entirely by electrical devices.





The VODER is a "modification and adaptation of electrical equipment designed for research in the characteristics of human speech. The equipment has two parts: The first analyzes telephone speech currents instantaneously and automatically; the second synthesizes speech currents in accordance with the results obtained by the analyzer."

After years of experimentation with mechanical gadgets made of wood, rubber and leather, of which paddles, levers and small resonator boxes were constructed to substitute for the various

positions of the tongue and the changes in the shape and size of the mouth cavity, Kempelen finally developed a working model with which he successfully demonstrated the mechanics of speech as represented by the various vowel and consonant elements.

With a keyboard of thirteen keys, made to operate the gadgets representing these individual elements, he was enabled to produce actual speech in German, French and Latin. He became so expert in the manipulation of this machine that he traveled throughout Europe demonstrating the performance of this apparatus to scientific and educational bodies.

In their final form, most of these gadgets were of such simple character that they serve today to illustrate the fundamental principles on which such mechanics are based.

FILM — THE HUMAN TONGUE

To illustrate the remarkable motility of the tongue I present another film which practically emphasizes these tongue activities. I have selected a number of young adults, most of them students in the Teachers Training College of Central Institute for the Deaf. Each subject offers an unusual and different tongue activity.

THE FLUTED TONGUE

This is an exaggeration of control and strength by practice of the instrinsic muscles of the tongue, especially the superficial transversalis muscles.

THE CRENATE TONGUE

Here we find a combination of bilateral intrinsic muscles representing both lateralis and transversus pairs and showing unusual control of these superficial muscles. As these pairs of bilaterally similar and limited to the central raphe, the resultant picture is the Crenate Form.

THE BROAD TONGUE

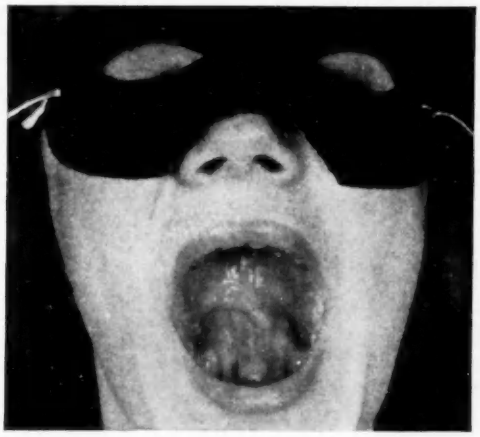
Relaxation of the intrinsic muscles, when carefully practiced, is as important an element in changing the shape of the surface of the tongue as is accomplished by contractability of these same muscles. In this act, all of the superficial muscle structures of the anterior third of the tongue are thoroughly relaxed.

THE NARROW TONGUE

Here is illustrated, in the same subject, the converse of the upper illustration. In the upper illustration all of the instrinsic muscles are relaxed; in the lower illustration the several pairs of intrinsic muscles are contracted, thus narrowing the entire anterior third of the tongue.

THE TIP OF THE TONGUE

In this act demonstrating tongue muscularity, the tip of the tongue is narrowed arched upwards and the anterior two-thirds of the tongue protruded to such a degree that the subject is able to touch the nose and even beyond the tip of the nose with the tip of the tongue.



THE TONGUE PLACED BEHIND THE SOFT PALATE

Placement of anterior half of tongue behind the soft palate and filling the nasopharyngeal cavity with this tongue mass is an unusual contortion of the tongue quite rarely seen.

The circumstances which actually induced this investigation are to be found in three cases of total amputation of the tongue, this radical surgery being necessitated by cancer of this organ.

Briefly, these cases may be described seriatim as follows:

Case 1. — C.C.M.F., male, age 59 years, was seen in July, 1931, with a very bad carcinoma on the left side of the tongue. A thorough left-sided neck dissection and complete removal of the tongue extending down to the cornu of the hyoid bone, was done by Doctor James Barrett Brown. The neck wound was left open and he was fed with a feeding tube for several weeks. He made an almost uneventful recovery, and the oral fistula from the mouth to the neck was closed some weeks later.

Since then, the patient has made a remarkable rehabilitation of his life, is able to carry on his work as a plumber, is easily understood by anyone and completely understood over the telephone. He states that he has the same sense of taste that he always had and requires the same seasoning in his food. He has been issued three insurance policies and at this date, October 12, 1939, he is apparently in perfect health with no evidence of any local or distant lesions.

Case 2. — W. S., male, age 64, was operated on January 15, 1937. His surgeon, Doctor E. Lawrence Keyes, reports as follows:

"Patient had sores on his tongue for about a year and a growth on his tongue for three months, quite painful when he chewed. Examination showed a large lesion over the anterior third of the dorsum of the tongue, measuring from 1.7x1.5 cm., involving chiefly the mid-portion and extending somewhat to the right. The margins were raised and pearly, and the growth was papillary in character. There was a second similar area on the right margin of the tongue somewhat smaller. On April 10, 1937, his tongue was amputated at the Jewish Hospital. All of the right half of the tongue and the anterior and middle thirds of the left half were removed purposely leaving a portion of the posterior third of the left side. The patient made an uneventful convalescence and since that time has shown no evidence of recurrence."

On February 18, 1939, Keyes demonstrated W. S.'s ability to speak with such a small residuum of tongue tissue, before the meeting of the National Forum on Deafness and Speech Pathology, in St. Louis. He was readily understood in the back of the room. His only defective speech elements were voiced and unvoiced *th*.

Case 3. — W. M., male, age about 71 years, operated for diffuse cancer of the tongue about thirty years ago by Doctor E. L. Bernays. The anterior tongue was radically removed, all muscle and glandular tissue being carefully dissected away. This patient made an uneventful recovery, has continued at his profession actively and is in good vigorous physical condition for a man of his age. His speech is understandable, fluent and seemingly produced without effort. This speech accomplishment is entirely the result of the patient's own efforts, and no speech coaching has been given at any time.

It is a singular fact that these three cases as well as others that have been discussed with surgical colleagues, have all been able, after their convalescence, to produce oral speech that is understandable and quite distinct as to its articulation nicety. In almost every case such speech was self-taught, and was accomplished without the assistance of especially trained teachers. These cases, then, present a challenge to those concerned with speech production, phonetics and articulation. If the tongue is so indispensable to speech and its production, how do these subjects substitute the

necessary accessories in the complicated mechanism of this function?

Finally, as a climax to this investigation, I present one of these subjects, Mr. C. C. M. Finn, who has kindly obliged me by offering himself as a practical example of a man who can comfortably and effectively continue his social contacts by speech minus a tongue.

For over a century the tongue has been considered as the most indispensable organ in the production of human speech and its position in the oral cavity for the enunciation of consonant elements has been classically considered and taught in all literature of speech and phonetics.

In the light of modern acoustic principles and mechanics of speech it is possible that much of this information concerning the physiological functions of the tongue may have been regarded with too much empiricism. This has been brought home to me most vividly in a series of recent clinical cases recorded and analyzed in the sound laboratories of C.I.D.

A most effective example to illustrate our exaggeration of the importance of tongue positions in the production of consonant elements of speech is offered in the phonograph record now presented of a case of total amputation of the tongue.

The patient, Clarence C. M. Finn, fifty-seven years of age, a plumber by trade and a resident of St. Louis County, was operated in July, 1931, by Doctor James Barrett Brown, of St. Louis. The operation was imperative and consisted of complete extirpation of the tongue for cancer. The tongue was amputated flush with the cornu of the hyoid bone and not a vestige of tongue tissue remains.

This patient made an uneventful recovery, has had no recurrence of this pathology in over seven years, and his rehabilitated speech is self-taught and was acquired within a few months after the healing of the operated field.

Before commenting further on this unusual speech rehabilitation I will ask Mr. Finn a few questions to indicate his facility in daily conversation:

Q.: What is your name?

A.: Clarence Cornelius Michael Finn.

Q.: Where do you live?

A.: I live at 201 East Etta Avenue, St. Louis County.

Q.: How old are you?

A.: I am fifty-seven years of age.

Q.: What is your occupation?

A.: I am a plumber.

Q.: How long have you been engaged in this work?

A.: ———— years.

Q.: Have you found the absence of your tongue a special handicap in carrying on your trade?

A.: No, I have no trouble in getting those with whom I come in contact to understand everything I say.

Q.: Did you receive any medical treatment for this pathology of the tongue before you consulted your surgeon?

A.: Yes, I was treated by a doctor who used what is called "cancer paste" for————months, and I want to add that it was one of the most painful experiences that I have ever had.

Q.: How soon after the amputation of your tongue did you find it possible to make yourself understood by speech?

A.: ————————

Now, Mr. Finn, I should like you to illustrate with your own voice and speech what facility and accuracy you have in fluent speech. For this purpose we have arranged a series of sentences in which initial consonants are used that have always been considered impossible without the tongue assuming certain positions in the mouth cavity. These consonant elements are offered to convince the listener of the ease, quality and distinctness of your speech.

Please read the sentences that have been prepared to illustrate the individual consonant elements. The first consonant I will ask you to illustrate is *T*.

First is the letter *T*.

The two tall travelers told the tale to Thomas
Ten and ten and two are twenty-two

Next is *L*.

Longfellow loved little children
Luke lost his luggage in the lake

Then comes *K*.

The cook cut the cocoanut cake into quarters
The cat climbed the tree and caught the canary

Now the letter *N*.

Ninety-nine names were nailed to the sign
No one knew the names of the nine new navy cruisers

The next letter is *D*.

Dan and David went on a daring adventure
Don't deny yourself the delight of your daily dozen

Then follows *G*.

Are you giving grandmother a Christmas gift?

Finally a sentence that contains all of the above consonant elements:

Good gracious! did I take the candy from little Nellie?

This demonstration offers practical evidence of ability to produce understandable speech involving the speech elements which heretofore were thought impossible without the aid of certain positions of the tongue in the mouth cavity.

For *T* we have always taught that the tip of the tongue must touch the posterior surface of the upper incisor teeth.

For *K* the middle third of the tongue is arched so that it approximates the arch of the palate above.

For *L* the tip of the tongue is raised and the anterior third of the tongue partly depressed in the floor of the mouth.

For *D* a similar position to that used for *T* is assumed.

N has a similar position but includes a different placement of the vocal element as it reaches the nasal resonating cavities.

Added to this tremendous handicap this patient has but three remaining teeth and much of the alveolar process of the jaw has been absorbed as is frequently found in the aged.

Finally, the lower branches of the facial nerve supplying the left upper and lower lips were cut during the operation; yet with the absence of these three important factors in the production of speech, this gentleman has given an excellent account of himself in the quality, distinctness, inflection and volume of spoken language.

Here is a real problem for research and perhaps reconstruction of our knowledge of speech physiology as it concerns the tongue.

In the study of the actual mechanics of speech it will be seen that, in the formation of the various consonant elements, the prin-

cipal role of the tongue is to intercept, direct or change the column of air, either simply a puff of breath or vocalized, as it is projected from the larynx to the mouth cavity.

In the case of tongueless subjects, who produce very creditable and understandable speech, and where the tongue has been completely removed, it will be observed that the fascia floor of the mouth moves actively and effectively to the positions in the oral cavity to simulate the movements of the tongue. Other accessories, such as the lips, lower jaw, soft palate, uvula and buccal muscles assume exaggerated positions to assist in this newly-established mechanics of articulation.

In practice we have frequently noted in glossitis, tumors, painful ulcerations and other pathologies, that the movements of the tongue are greatly impeded and sometimes entirely checked, the tongue then acting in the mouth cavity as an unyielding foreign body, unable to carry out its ascribed function of regulating and distributing breath and voice sounds sent through the glottis.

The observations and successful experiments of Kempelen, Kratzenstein, Paget and others are additional evidences that the tongue is not an indispensable organ of articulate speech.

These observations lead to the conclusion that the empirical teachings concerning the physiology of the tongue, as we have accepted them for over a century and the evidences presented by tongueless patients with understandable speech, call for a revision of the mechanics involved in articulation and an analysis of the actual factors functioning in this important act of speech.

Mouth and tongue are put in movement with each voice vibration, either sibilant or vocalized, and each column of air is guided in different directions in its exit from the mouth, producing different sized and variously formed openings and thus forming the necessary variations to produce both vowel and consonant elements.

As gathered from a careful study of speech mechanics, as offered by Kempelen in the construction and performance of his speech machine, such mechanism can be measured with fairly accurate mathematical certainty. Guided by this principle we continue the analysis of speech sounds as applied to the tongue and all accessory organs of the mouth and offer a working project of what substitutions can be made when the tongue is removed as an important functioning factor.

In our observations of the mechanics of the human body we have noted many compensatory changes that reconstruct functions

taking place after the loss of apparently indispensable organs. Thus we have:

(a) Voice produced by exaggerated action of the ventricular bands (false cords) when the corda vocalis (vocal cords) have been destroyed.

(b) Anastomosis of blood vessels when an arterial supply has been blocked by injury or disease.

(c) Reconstruction of speech and hearing centers in the cortex when normal functioning areas have been impaired.

(d) Compensatory acts of the kidneys, lung, heart and spleen when pathologies affect these important organs.

(e) Replacement of biochemical action when glandular structures offer insufficient or over-produced supply of their secretions.

(f) The vast problem of allergies, its tests, reactions and restitutions, is a concrete example of chemical substitution.

Whether such compensations are affected by chemical, mechanical, surgical or other means, the fact remains that substitution of functions is a profound study of the future; and the tongue and its relation to the actual production of speech is no exception to this generally accepted law in the continuance of physiological functions.

It is this compensatory functional activity of the mouth and its accessories that has stimulated my thought to determine the mechanics by which speech may be re-established after the removal of the tongue.

From the mechanical principles here analyzed and from actual observations on tongueless patients, we feel justified in the deduction that the fundamental physics involved in the effects of a column of breath or vocalized air in the production of speech are:

1. Compression and control of the air column as it enters the oral cavity from the glottis.
2. Change of direction of this vocalized air as it is formulated into articulate speech elements.
3. Modification in the shape and size of the elastic oral cavity to facilitate the construction and production of both vowel and consonant speech elements.

The mechanics of articulate speech, therefore, involve not only the tongue but also the other accessories of the mobile oral cavity, comprising the fascia of the floor of the mouth, the lips, the teeth, the soft palate and the buccal areas.

In the teaching of speech, especially to the deaf child, and in the correction of defects in speech, less attention should be given to empirically prescribed positions of the tongue than to the modification of shape and size of these accessories of the oral cavity, and to breathing exercises and breath control.

In the finesse of production of the singing voice, breath control and voice placement have long been recognized as of major value, and the same philosophy and practice are logical conclusions where the speaking voice is concerned.

The analysis which I have endeavored to present in this thesis offers, I think, sufficient evidence: (1) that the tongue is not absolutely indispensable as an organ of articulate speech; (2) that various other parts of the mouth cavity may be utilized to replace it; (3) that understandable, fluent speech can be produced without a tongue; (4) that our knowledge of the physiology of the tongue as an organ of speech is subject to much needed revision.

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THE EFFECTS OF TOBACCO (SMOKING) ON THE RESPIRATORY TRACT

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IT IS WITH considerable reluctance that I acceded to our Secretary's request to talk to you about the effect of tobacco smoke on the membrane of the upper respiratory tract, and this for the very excellent reason that I know practically nothing about it. It was pointed out that if after working for upwards of two years on the subject one still knew nothing about it, it might be interesting to state such a fact, in view of some of the glib opinions that have been expressed in regard to tobacco smoking. It might also be of interest to show a few of the methods by which we have gone about trying to find out something about tobacco smoking. With this introduction I merely wish to emphasize the fact that that is what I am doing. I am not telling you much about the results because I do not know them.

My impression, after a long series of observations, is that the average individual, in fact the great majority of people, can smoke without any appreciable effect on their upper respiratory tracts. There is a small percentage of people who cannot smoke at all, just as there are a few who cannot eat strawberries and for very much the same reason. Then also there is a large percentage of people who cannot smoke as much as they want to. When a patient comes to one and says, "Doctor, will it do me any harm if I smoke?" he usually means, "Will it do me any harm if I smoke excessively or if I eat my cigar?"

In the first place the first problem with which one is confronted in studying cigarette smoke is the fact that it is so extremely variable. When one wants to study a product he has at least first to put his finger on the product. Cigarette smoke is a combination of tarry substances and volatile gases, carbon dioxide, and also the vapors of certain aromatic substances in the tobacco. The part that you see is composed entirely of tar in very microscopic globules. The combination of these substances and, in fact, their

very chemical composition, depends upon a great number of factors. It depends particularly upon the temperature of combustion, which in turn is dependent upon the way in which the cigarette is smoked. If you hold your cigarette like this (with the lighted end upwards) it burns a great deal slower than if you hold it like this (downwards). If you puff it rapidly it burns fast and it burns hot. If there is a long time between puffs it burns cold. If the air is dry it burns faster. The character of the tobacco further influences it.

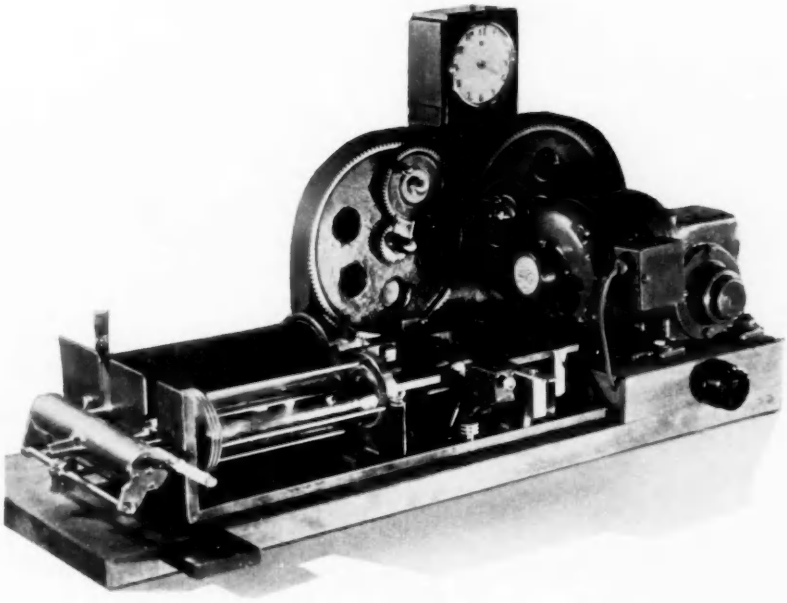
So to begin with one has such a terrifically variable substance with which to work that it was absolutely necessary to devise some method by which smoke could be produced which was comparable with some other smoke which might be produced six months later. In the first place the tar that you see settles down very slowly, so that the effect on the throat or upon any membrane is a very minute one at any given time. If you take a puff of cigarette smoke and blow it into a small box lined with white paper it has to stay there for upwards of five minutes before the paper is stained at all. If you open the box before that time has elapsed the paper remains perfectly white. Of course, nothing like that length of time expires in a puff with a cigarette. So that the very minutest amount of that tar is deposited upon the membranes of the mouth and throat.

We experimented for a long time with machines designed to puff cigarettes at intervals and turn out standard smoke. We found that we had to do away with anything that had rubber valves in it, water valves, mercury valves, or any adjustable valves. We had to dispense with any machine that worked delicately, and finally evolved a machine that weighs about 100 pounds, that has very heavy cog wheels and motor and automobile piston, and similarly heavy things, so that it would grind away at its usual rate and give us a standard smoke.

(A motion picture of the machine in operation was shown.)

The cogs function to make the cylinders work every thirty seconds. There is a large cylinder representing the capacity of the lung, 500 cc., tidal air and a small cylinder representing the capacity of the mouth. Every thirty seconds the small cylinder takes a puff from a cigarette, 35 cc., mixes it with the 500 cc. of the lung, and blows it out of a tube for experimentation. This little balloon on the left represents the mouthful. We put the balloon in to keep the glass cylinder from being gummed up with tar. The valve on the left end works entirely mechanically. The cigarette on the lower left will give you some idea of the size of the instrument.

The smoke is brought into a box and fed to a rabbit. He takes it very much in the concentration that the human nose gets it, and as soon as he has had his breath an air current clears the box so that he is not continually smoking. There is an interval of thirty seconds before he gets another smoke. I might say for the comfort of some of you heavy smokers that one or two of these rabbits hated this procedure pretty badly. Most of them did not seem to mind it, but one of them actually loved it. When we put the rabbit near the machine he went right in and put his nose on the place where the smoke came out and stayed there.



After exposing a considerable series of these rabbits for a period of something like six weeks, we then killed the rabbits, looked at their membranes — the upper and lower respiratory tract — and found no change whatever that we could demonstrate; not the slightest difference between these rabbits and the controls.

We had to devise some means also for getting hold of the tar so that we could apply it in some sort of standard way to a human throat. It is going to have to be done in a large series of cases. We have already made something short of 1,300 observations only to conclude that routine human observation of the throat is not

very reliable. We have devised a colorimeter which will measure accurately the color changes in the throat because we do not know for what we are looking. We do not know that the initial change may not be a paling instead of a reddening. There may be an astringent effect first or last. At any rate there are certain effects that we have noted that we can put to use. For instance, we will make use of what is known as the impingement effect to collect the tar from the cigarette. I will show you how it works.

If we blow the smoke from a cigarette through a glass tube, against a sheet of paper, practically none of it sticks, as I told you before in regard to the little white paper box. However, if we blow it with some force through a capillary tip, so that eddies are formed, the tar is forced against the paper more or less forcibly and sticks there and can be gathered. That is the principle, of course, utilized in the cigarette holders employing a cigarette as a filter. It is simply a series of capillary tubes.

However, we have rigged up a contraption of glass tubes, so that the smoke which we are studying can be deposited right on the tip of a glass rod. We can pile up the tar and gather the fumes in balloons and apply them to the throats of human subjects. As I said before, there are many variable factors that control the color changes in the throat, the temperature of the room, the condition of the climate, the patient's condition of health, the patient's recent eating of a meal, the patient's having a cold, and what not, so that literally thousands of these tests will have to be made before we can arrive at anything of scientific value.

If these very few short descriptions of a laborious job are of any value at all, it is probably to point out that the problem is an involved one and that one must investigate the source of any statements made about tobacco smoking before giving them any credence.

INNOCUOUS OILS USEFUL IN RHINOLOGIC PRACTICE IN CONTRAST TO THE USE OF MINERAL OILS

FRANK J. NOVAK, JR., M.D.
CHICAGO

IT IS OF INTEREST historically that the first use of mineral oil was for medicinal purposes. Samuel Martin Kier was the first refiner of oil. He first refined oil, not for lamps, but for medicine a hundred years ago. When the oil springs of Pennsylvania were discovered, oil was skimmed off the water and the product was used in a variety of "remedies." Quoting from an editorial in the Chicago *Daily News* which appeared on the 125th anniversary of Samuel Kier's birth:

"Kier's method of refining, the basis of most refining ever since, came naturally from the western Pennsylvania environment. Whisky was the great staple, not second even to pig iron in that region, and nearly every farmer had a still. Kier devised a special one-barrel still that derived something very much like modern kerosene from Pennsylvania crude, and after his medicine ventures had failed to meet expectations, he turned to illuminating oil.

"Kier never attempted to patent his refining inventions. The American West in those days was full of his kind, men who were only secondarily interested in the money to be made out of 'improvements.'"

The attention of people in general has been called to the possible deleterious results from uncontrolled self medication with nose drops. The intelligent public which in these days is well informed about things medical is keenly alert to the warnings that have been disseminated through the press and by radio regarding the indiscriminate use of oil in the nose. In view of this reaction it may be well to examine the evidence critically to see how much we really know about some of the medicaments that are used intranasally.

There is ample clinical and experimental evidence to show that the mineral oils are profoundly irritating to the alveoli of the lung both in the experimental animal, and in the human. Laughlen in 1925 described the lung pathology resulting from the aspiration of mineral oil. Since then numerous writers have called attention to the fact that not only in infants but also in the adult is there an increasing incidence of lung pathology due to oil aspiration. All of this is incontrovertible. Cannon and Walsh have shown by extensive and conclusive experimental work that:

1. In the rabbit when small doses of mineral oil are instilled into the nose the oil finds its way almost immediately into the lung.

2. This is followed promptly by an increased premeability of the alveolar wall, and the exudation of albumin.

3. With the lapse of time further extensive changes in the lung occur which have been designated by various writers as lipoid pneumonia, lung paraffinoma, etc. Cannon and Walsh say, "We wished to ascertain the early effects upon the lungs, particularly within the first few days, of medicated oils introduced into the nostrils of normal, "snuffles-free" rabbits. The oil was given intranasally by means of a tuberculin syringe without a needle, in doses in most instances of 1 cc., 0.5 cc. per nostril. The rabbits were usually in a sitting position in a rabbit box, and under these circumstances the oil almost invariably gravitated to the anterior pulmonary lobes. The rabbits were killed in two or three days after instillation of the oil, the lungs fixed *in situ* with Zenker-Formol solution, after which celloidin sections were prepared and stained with hematoxylin and eosin. The following medicaments were used: plain mineral oil, Vick's Vatronol, Mistol, Pinoleum, benzedrine in oil, Chloretone inhalant and Oliodin. The changes observed in the lungs were essentially similar for all preparations and were as follows: Within twenty-four hours there was a focal edema of the involved portions of lung, with extensive desquamation of septal cells, with many filled with large oil vacuoles. The alveolar walls were thickened and in such areas there was definite focal atelectasis. In some instances there was associated acute bronchitis, although usually this was absent. The histologic picture was constant, and showed definitely that such medicated oils and even plain mineral oil are toxic enough to pulmonary tissue to cause increased permeability of capillaries and leakage of albuminous fluid into the air spaces.

In the light of this experimental proof of the great toxicity of mineral oil to the lung tissues, and the clinical evidence of lung damage from mineral oil, is there any justification for the use of oil in nasal therapy?

The inquiry resolves itself into three parts. First, a consideration of the therapeutic value of oil, and the effects on the soft tissues of the nose, normal and diseased. Second, what is the evidence that oil finds its way into the lung of the human as readily as it does in the rabbit? Third, a consideration of the relative irritant properties of the various oils, vegetable, animal, and mineral.

Despite the admirable work of Lierle, Proetz, Moore, Hilding and others it should not be assumed that the final and complete story of ciliary activity and other nasal physiology has been told in its entirety. No one has as yet seen the effect of oil, or for that matter of any other substance directly, through the microscope upon normal ciliated epithelium, in its normal surroundings within the nose. The behavior of ciliated epithelium treated with various substances in surgical exposure of the epithelium or again in tissue culture may be very different from the behavior of the epithelium in its normal undisurbed habitat. The ciliated epithelium undergoes a multiplicity of chemical and histological changes in its departures from the normal, and what the precise effects of various substances may be on these changes is totally unknown. Fox has observed that intranasal spraying with mineral oil produced desquamation of surface epithelium when continued for nine months. It would be faulty reasoning to conclude therefore that spraying the same oil into the human nose for a few days has a deleterious effect on the surface epithelium.

Proetz, Lierle, Moore, and Hilding have studied the effect of oil, and oil as a vehicle for drugs on ciliary action. They have observed the effect of oil on mucous streaming, and in general have found that oil slows up or stops the movement of mucous blanket. From these observations the following conclusion only is warranted, namely that in a normal nose with a normal mucous blanket oil interferes with the normal mobility of the mucous blanket. To conclude that oil is a deleterious agent is faulty reasoning. Oil as a therapeutic agent is not used in normal noses. What of the nose in which there is no fluid mucous blanket? Or the one with perverted glandular (mucous) secretion, or the one with abnormal ciliary activity? What experimental or clinical evidence is there that oil under these conditions is a deleterious therapeutic

agent? In view of the fact that our knowledge is so incomplete and fragmentary a categorical statement that oil in the nose is worthless, or even a deleterious therapeutic agent, is open to some question.

Walsh reaches the following conclusion, "In view of the fact that the maintenance of the physiological functioning of the nasal mucous membrane and the promotion of adequate drainage from the nasal sinuses are the two essentials in the treatment of upper respiratory infections weak saline solutions of ephedrin and neosynephrin appear to be the medications of choice for intranasal application." This is a sound conclusion if one does not read into it more than it says.

There are pathological conditions in which there is no vestige of normal physiological function in the mucous membrane, and there is as yet no experimental or other proof that oil is of no value, or that it is deleterious in its action in such cases. The fact that it is gratefully received by the patient may not be without importance.

In general one may say that there is not enough precise experimental evidence, nor is there enough valid clinical observation from which there is justification for any categorical conclusion regarding oil as a therapeutic agent in rhinology.

Now in view of what happens to a rabbit's lung when mineral oil is dropped into the rabbit's nose, is the use of mineral oil in the human nose a justifiable procedure? It would appear that there is none. This viewpoint gathers further weight from the fact that there are wide differences of opinion among rhinologists regarding the actual therapeutic value of oil in the nose.

However that may be, certain significant facts should not be lost sight of. Cannon and Walsh quote Ikeda who found 106 cases of oil aspiration pneumonia reported by 24 authors in the ten years from 1925-1936. Now, among the laity the use of oil drops for the nose is one of the three most common types of self medication; the other two being the use of cathartics, and of aspirin. This implies the consumption of large amounts of oil by an enormous number of persons, and yet over a ten year period there are only 106 reported cases of lipoid pneumonia. Granting that many such are undiagnosed, still the disparity between the number of reported cases and the huge number of individuals who make it a regular practice of taking oil drops calls for some explanation. In all probability oil finds it way to the lung in the human being when instilled into the nose under exceptional conditions only. At this point it

may be well to point out that aspiration lipoid pneumonia has occurred in adults who have taken mineral oil by mouth for constipation. It is to be noted, however, that most of these individuals took the oil in large amounts over very long periods of time, and moreover some of them were disphagic from one cause or another. These same people were in greater danger of lipoid pneumonia from drinking cream or taking cod liver oil, because both of these are much more drastic irritants to the lung than is mineral oil.

The animal oils such as lard, cream and cod liver oil have the greatest necrotizing action on lung tissue. The mineral oils rank next as irritants. The least irritating are the vegetable oils such as cotton seed oil, sesame oil, and olive oil. The relative toxicity of the three kinds of oils was determined by Pickerton and Ikeda. The toxicity is dependent upon the content of free fatty acids, and the vegetable oils contain the least free fatty acids.

CONCLUSIONS

The use of any medication in the nose excepting ephedrin in normal saline is hazardous in some individuals. According to Ikeda no type of nasal oil should be used in persons with tongue lesions, lesions of the pharynx, hemiplegia "or some other neurogenic disturbance." What applies to oil applies with equal force to practically all of the commonly employed drugs whether in oily or aqeous vehicles. Furthermore, infants and debilitated children and the aged are more liable to aspirate fluids taken either into the nose, or orally, and in these the use of mineral oil, or any other kind of oil is clearly contraindicated. However, the use of oil when indicated, with care and judgment in the rhinologist's office, may not be criticized in view of the fact that legitimate differences of opinion exist regarding its therapeutic efficacy. It is ,however, safer to use vegetable oils in perference to mineral oil. The indiscriminate self medication with various oils by the laity should be condemned.

DISCUSSION

T. E. Walsh, M.D., Chicago: Dr. Novak has given us a very excellent presentation of the problem of the use of oils in nasal therapy. He asks us three questions: First, what is the therapeutic value of oil in the nose? Second, what evidence is there that oil used in the nose reaches the lungs in humans as easily as it does in animals? Third, what are the relative values of vegetable, animal, and mineral oils? I would like to consider these questions in this discussion.

Proetz, Moore, Hilding, Lierle and others have shown in the living animal that mineral oil when placed in the nasal membrane does of itself no harm to the activity of the cilia, but does embarrass their function by lying inert on the mucous blanket that covers them and so by its very weight gives them more work to do. They have also shown that mineral oil at least is a very poor mixer with the mucous that covers the epithelium. It would seem, therefore, that this oil is contraindicated as a vehicle for drugs that we expect to act as a vehicle on the epithelium lying beneath the mucous membrane.

Before any drug is used in the nose it is well for the rhinologist to ask himself several questions. For instance, does this drug do what I expect it to do in the nose, for example, in the case of antiseptics? Is it truly antiseptic? Is it bacteriacidal? If it is, can it perform its function without damage to the tissues that it is supposed to help? Does the drug I am about to use do any harm to the tissues in the nose that it is supposed to help, or if it does not, does it do any harm elsewhere in the body? And, finally, is there any good reason I should use that drug?

If one considers mineral oil at least in the light of these questions one has to come to the conclusion that it is contraindicated as a nasal therapeutic agent because it embarrasses the ciliary function and instead of helping the membrane to return to its normal state, it hinders it, and finally because it does without a shadow of a doubt reach the lungs when placed in the nose in some cases.

Laughlen in 1925 first described this condition called "lipoid pneumonia." He found in the lungs of children areas of concentration in which alveoli were filled with macrophages containing oil. Later, if the condition was of long standing, there was fibrosis present. Although nasal oils had been used for years before this, it was not until 1925 when he first described this condition and since his original report that more and more cases appeared in the literature. More work has been done by Pickerton, Cannon and others, and this disparity in numbers between the cases of lipoid pneumonia and people using oil may possibly be due rather to the failure of the pathologist to recognize lipoid pneumonia at autopsy than to the fact that the oil has not reached the lungs. One might here draw an analogy between lipoid pneumonia in the use of nasal oil drops and alcoholic cirrhosis in the use of alcohol. Most of us use alcohol but I doubt if we see a great deal of alcohol cirrhosis at autopsy, but no one can gainsay the fact that alcohol is a factor in alcoholic cirrhosis.

If mineral oil and other oils reach the lungs in humans it is possible for other medication to do so, and it is important in this consideration to think of the damage done to the lung tissue by such medication as Neosilvol or Argyrol, particularly in infants. When these medications are poured down the nose of an infant when they reach the lungs they carry infection from the nose with them, and it is possible that in a great many of these children, small infants, who develop bronchial pneumonia following colds, the actual nasal medication may be responsible for the etiology of their bronchopneumonia and remain unrecognized. If oils must be used in the nose — and they must be under some conditions, such as atrophic rhinitis — then we should choose the least damaging of the oils, such as vegetable oils in preference to any other.

W. E. Grove, M.D., Milwaukee: Oil, as a vehicle for intranasal medication, has been used for a long time. That this oil per se can be deterimental to the tissues of the lower respiratory tract has only become apparent in the last decade or so by the reports of lipoid pneumonia or pneumonitis, which have begun to filter into the literature. The essayist has called attention to the relative irritability of mineral, animal and vegetable oils. He has said little about another phase of the question, which to my mind requires elucidation: the relative absorption of these various oils. Is the resulting pneumonitis caused by the immediate response of tissue to an irritant or to the fact that the oil remains unabsorbed in situ for a long period of time?

That an irritating and non-absorbable oil does produce pulmonary changes, even in the human, is well established. That it does so more frequently than the 100 cases reported in the literature in the last ten years is unquestionable. The reported number of cases is only an index of the alertness and intelligence of the physician and not of the incidence of the condition, but to lay this situation entirely at the door of the self-medicating public or the manufacturer is not quite just. In prescribing the use of oily nose drops the entire professoin, and particularly the pediatrician and otolaryngologist, is also to blame. The less irritating vegetable oil can be substituted for the hydrocarbon oil in the nose drops, but even so the use of the mineral oil in the hands of the discriminating physician should not be utterly condemned. The essayist has indicated the crux of the whole matter in the expression "injudicious and indiscriminate use of."

If the otolaryngologist wishes to use an oil, even a hydrocarbon oil, judiciously in a finely divided state in a nebulizer, I cannot convince myself that it is dangerous or detrimental either to the ciliated mucosa or to the lung tissues. Quite different is the use of this medication in the form of nose drops and more especially while in the recumbent position. If such medication is prescribed for home use it should be in a finely nebulized form and all such medication should cease at least one hour before bed time.

A. G. has been under my observation since 1920. His turbinates had been previously resected because of nasal obstruction. I had repeatedly examined his sinuses and always found them negative. In July, 1938, he was referred by an internist for sinus examination because of easy fatigability, a twenty-pound weight loss, and a persistent cough. The sinuses were again clear. At this time he volunteered the information that for several years he had been in the habit of flooding his nose with Mistol by means of a coarse spray just before retiring and while in a recumbent position. Physical examination and x-rays of his chest revealed what was interpreted by internist and roentgenologist as a lipoid pneumonitis. After stopping the use of the Mistol he rapidly regained his weight and strength and his cough gradually disappeared. The lung appearance, however, remained unchanged as an x-ray taken one year later will show.

Frank J. Novak, Jr., in closing: I wish to thank the gentlemen who have discussed this paper. I do not wish to enter into further discussion, except on one point. Doctor Grove mentioned the fact that there is a difference in

the absorbability of oils when they reach the alveoli of the lungs. As a matter of fact the things that happen in the alveoli of the lungs are these: Mineral oils in particular have a certain potentiality of stimulating the microphages which are found in the alveolar wall, so-called dust cells, and these cells take up the oil in large droplets. The vegetable oils also do this, but to a minor degree, and the vegetable oils have a tendency to lie free in the alveoli for a time and may be expelled by the act of coughing.

I wish to suggest that all of you see the exhibit of Doctor Walsh in the scientific exhibits showing the experimental work that has been done on this particular subject.

THE EPIPHARYNX — THE ALMOST UNKNOWN IN OTOLARYNGOLOGY

T. E. Carmody, M.D.
DENVER

The epipharynx has been studied extensively by the embryologist and anatomist, because of its close association with the development of hypophysis eustachian tubes, sphenoidal sinus, tongue, palate and larynx, as well as the nasal cavities, and the pharynx.

While the facts elicited by these investigations are very important and interesting in laying the foundation for pathological changes in infancy or later life, it is impossible to go into detail in the time allotted for this paper. Furthermore, all the material has been correlated in previous late publications by Dorrance and others, which may be read with much benefit by anyone interested in the subject.

This region, because of its close association with the other organs of respiration, deglutition, phonation and hearing, is of especial interest to the otolaryngologist, and it is not given sufficient weight in otolaryngological examination as is evidenced by the fact that many publications cite pathological conditions overlooked by otherwise competent observers.

That examination is not easy must be admitted even in adults. Examination of children without anaesthetization is out of the question except in very rare cases. Indirect examination with tongue depressor and mirror is unsatisfactory in the majority of cases while the use of the Holmes Pharyngoscope presents difficulties not easily overcome in most patients.

The pharyngeal speculum of Yankauer is the most practical, although many find a palate retractor, Hasslinger or others, and mirror more to their liking in adults or anaesthetized children. The Kelly adenotome is a valuable instrument because of the fact that this area can be in view during removal. Why these instruments are not more universally used is probably because they are not thoroughly appreciated and have not been tried.

In patients who, for any reason, have lost portions of the turbinal bodies, direct view of a limited area may be obtained but insufficient, as a rule, so that other means are necessary to make examination complete. The study of the anatomical structures made in cases where the soft palate is lost by infectious disease or malignant growths, is comparatively easy. However, the loss from syphilis is rare today. In failure of development as in cleft palate, easy access for examination and treatment may be obtained.

The most frequent pathological change is hypertrophy of adenoid tissue which is normally found in the vault. In the embryo and in the infant the relationship of this cavity to the pharynx and larynx is important as pointed out by Ingersol. The distance between the vault of the pharynx and larynx is so very short in the foetus and changes very slowly in the first year after birth that the adenoid tissue may be found to extend almost into the larynx.

The epipharynx is of importance to us as the communicating passage between the upper and the lower respiratory tracts and the further communication with the middle ear. The linking with and continuation of this cavity with these other parts of the respiratory tract (for the middle ear and mastoid cells must be considered part of the respiratory tract) gives opportunity for pathological conditions of these communicating parts to be transferred by continuity of tissue. Because of the close association of this cavity with the ear, pharynx and larynx, we must be constantly on our guard. Not infrequently do we find the patient complaining that the first symptom of a cold is soreness in the epipharynx. From this point we may then have symptoms referred to either the upper or the lower respiratory tract.

Before any patient is anesthetized, either with a local or a general anaesthetic, we all examine very carefully the nose, mouth and pharynx, but I find that we frequently overlook the epipharynx. Furthermore, we find that cultures from the epipharynx so frequently give us the offending organism when it may not be found in the nose or pharynx. For a number of years we have been taking cultures from the epipharynx rather than from the other cavities because we find the offending organism in greater profusion even if found in the nose or pharynx.

Most surgeons at present are very careful in their examinations before any prolonged anesthetic is given, and it has been noted by Alexander and others that infection of the lung is found more often if the recovery from anesthetic is prolonged. We have, for

many years, placed our patients upon their sides or faces after they have been returned to bed, and in a few cases, have used the Trendelenberg position. However, Alexander advises this latter in all cases where it can be used.

The first thought, when we speak of infection from this cavity, is the middle ear, and the eustachian tube is supposed to be the link in the chain. When we take into consideration the possibility of a large adenoid in the vault, and the prolongations into Rosenmuller's fossa, there is good reason for infection of the mouth of the tube, or for pressure closing the tube. In either case, circulation is interferred with, both in the lining mucosa and the surrounding tissue. Most of us believe that infection travels through the tube because we find middle ear infection in babies and young children who are left lying on their backs, and in adults so frequently after forcibly blowing the nose.

The fact that the tube is short and fairly large in childhood may be the reason Dean and his co-workers, however, have found many bacteria in the surrounding tissue, which leads them to the conclusion that the route may be through the tissues outside the mucous lining of the tube. It makes very little difference by which route infection takes place if we are not vigilant enough to prevent its occurrence.

In speaking of infection of the middle ear, we are not limiting discussion to suppurative disease, for this is probably not the greatest cause of pathological changes which produces deafness, but may be the most frequent cause of death.

E. L. Jones has given us a number of cases covering this and other phases, and Yearsley contributed an exhaustive paper covering the whole subject as well as the ear. He makes a particular point of the interference with circulation in this region and its effect on the mental processes of the patient with enlarged adenoid which has been noted by many authors.

Often we have found drainage in the oropharynx although we found nothing in the nose or sinuses, but, on close examination, we discovered the pathology in the epipharynx or the posterior portion of the nasal cavity which could not be seen by anterior rhinoscopy. This I have seen on many occasions where it had been overlooked on previous examination by other careful examiners, or by myself.

Our predecessors found enlarged posterior tips of inferior turbinals which they cauterized or removed, and their success indi-

cates that these were not always due to sinus disease. Thickening of the posterior septum can be attributed to previous epipharyngeal disease. Sphenoidal sinus infection is probably very often found on post-nasal examination when we are unable to make the diagnosis in any other way. Furthermore, its cause may be previous epipharyngeal infection.

The most frequent growth in this locality is the lymphoid tissue which we designate as adenoid. Meyer of Copenhagen is responsible for our first interest in this tissue, but time will not permit our going into the history of this discovery. The fact that this is the most frequent operation together with the removal of tonsils, and that this operation is performed not only by many trained operators, but also by those who have received little or no instruction in the embryological or pathological development, or in the technique of removal, is a reason for our diligence in examining this cavity in all cases both before and after removal. Not only the remains of adenoid vegetation but the scarring around the eustachian opening, or the removal of part of the pharyngeal end of the eustachian tube, have frequently been noted. Leland first called attention to adhesions around the eustachian tube and these may be a frequent cause of abnormal action of the pharyngeal end of this organ. This condition has been seized upon by irregulars to promote a so-called cure of deafness. The failure to remove part of the central portion may leave a recess which may be, or is frequently, called Thornwald's Bursa. This condition is often overlooked. The danger of removing the bony prominence of the vestigial bodies must be thought of in too vigorous use of any instrument in this region. Also injury of the palate has been seen a number of times, apparently caused by vigorous over-use or maluse of instruments.

It is unnecessary to try to instruct this audience in the technique of the operation. However, the numerous cases seen where only the medium lobe has been removed, and sometimes this very incompletely, leads us to believe that this is one of the most poorly performed surgical procedures.

If we are to accomplish anything in preventive medicine, we must be on the alert in our examination of all patients, both before and after operation. While this is very important in the adult, it is of much greater importance during infancy and childhood because of the possibility of lower respiratory infection, and of the effect upon the hearing organ. With the large proportion of

our population suffering from defective hearing, it behooves us to use all diligence in removal of pathological tissue which may later be the cause of suffering, both physical and mental.

The fact that so few cases are found in which there is progress of pathological conditions after operation is a tribute to the immunity of tissues and not to surgery. That is poor surgery. If we had, as someone has said, all good surgery and all good medicine, we would have fewer troubles. The lymphoid tissue found in the vault and in the orapharynx is so often the cause of painful sensations and of cough not otherwise accounted for, that it deserves our utmost attention. The diagnosis of presence of adenoid growth is most frequently made because of mouth breathing. This may be due to several different causes: first, to enlargement of the tissue primarily present in the infant, as there is then a vicious circle producing the mouth breathing, the continuation of which produces narrowing of the nasal passages; second, narrowing of the oral cavity which, in turn, because of muscular action, produces further narrowing of the nasal passages. The effect on the teeth, both those present and those which may later erupt, produce almost untold injury to the whole organism. Some authors make a point of artificial feeding as a cause for adenoid enlargement which they think does not occur so often in breast fed babies. They might well also include thumb sucking because of the fact that it produces deformity of the palatal arch which, in turn, interferes with nasal respiration.

As to the results on the lower respiratory organs producing bronchitis and other pathological changes, time will not permit complete discussion. The secretion from the adenoids, be it either mucous or pus, is much more damaging in infants and young children than in adults because of the close proximity of the tissue behind the palate to the larynx, and through this to the trachea and bronchi. Furthermore, the swallowing of this secretion, especially at night, may cause severe derangement of the gastrointestinal tract. Yearsley has called special attention to this fact.

The tumors of this region may be either benign or malignant. The benign tumors are usually easily diagnosed because they have grown to considerable size before the patient realizes that they have difficulty in breathing. Those that originate in the nasal cavity or sinuses are usually diagnosed early. Malignant tumors, however, are insidious in their onset, and may have progressed to such an extent as to be inoperable and incurable, before diagnosis

is made. Lederer has recently reported a case of this nature in which the tumor was later engrafted on the palate. In these malignant cases the patient usually complains of difficulty in hearing or otalgia, and not of difficult breathing as is more frequently the case in benign tumors.

It is manifestly impossible to give more than an outline of the pathological processes which may involve the epipharynx. However, it is my hope that this will stimulate more frequent and more thorough examination of the crossroads of the respiratory and digestive tract.

DISCUSSION

LYMAN G. RICHARDS, M.D., Boston: I find that I am quite as guilty as possibly some of you may be in my neglect of this important, unknown region which Doctor Carmody has described. In searching my heart as to the reason for this I have to plead guilty, I think, primarily to the difficulty encountered in examining this region. I think that textbooks give a distressingly inadequate idea of such difficulties. How many times we see in a book a picture of a nasopharyngeal mirror, often of the size that we commonly use in the larynx and on it a complete picture of the whole nasopharyngeal region including middle, superior and inferior turbinates, the septum and both the eustachian tube and the adenoids. At times we learn that is far from the case, as you well know. It must be examined only by fitting of the mirror and an effort made to catch fleeting glimpses of several portions of this region.

Not only that but there is, I think, a very profound effect resulting from the position of the palate with reference to the posterior pharyngeal wall. There are unquestionably many cases in which this space is adequate for the insertion of a mirror. On the other hand we frequently encounter cases in which the soft palate hangs only a few millimeters anteriorly to the posterior pharyngeal wall, in which with even the best relaxation it is impossible to insert a mirror in this region. The Yankauer speculum undoubtedly fills a breach in this connection. Adequate anesthesia is certainly necessary for its use in most patients. I think it could be improved if it were equipped with a distal light in the same manner as the ordinary laryngoscope. A suggestion I heard today was that it be made with a darkened inner surface to prevent the reflection of light against the side walls.

I think that Doctor Carmody would be interested if he got the reaction that I did today of the frequency now with which men are taking an interest in this region and apparently are determined to examine it more thoroughly. They are dusting off their nasal pharyngoscopes and their Yankauer speculums and are apparently stimulated, for the reason it may be, to give this region a more thorough examination. It certainly is an important region to examine and I think we have all been somewhat negligent in the past, at least I know I have, in examining this area.

I am concerned somewhat with the question of these adenoid rests and the scar tissue and the occlusions about which we hear so much in this region, and I sometimes wonder if it may not arise in connection with the still almost universal removal of adeniod tissue in those instances in which there does not seem to be any specific clinical indication for it. By that I mean the routine supplementary adenoidectomy in connection with tonsillectomy particularly in children. I have asked colleagues, when looking over their shoulders when they were operating, why they took the adenoids out when they proved to be only a mere fragment of tissue, or at least a small amount, there being nothing to suggest any involvement of the eustachian tubes and no evidence of nasal obstruction, and they look at me in blank amazement at such a question. I think that is a fair question to ask: Why the routine removal of a small amount of adenoid? Certainly time and again, in adult tonsillectomy, an examination of the nasopharynx reveals that region formerly occupied by the adenoid to be as smooth and as non-pathological as anyone could imagine.

This leads me to wonder about the question of the pharynx in respect to lymphoid tissue; whether it has some effect that may be important and whether we are not perhaps encroaching at times upon an area that had better be left alone. That sounds like otolaryngeal heresy. Certainly, you see many patients with an irritated pharyngeal region and with posterior bands and lymphoidal nodules scattered over the pharyngeal wall. That has been a problem for all of us to explain and to treat adequately these returns or rests or recurrences of lymphoid tissue and adenoids. It gives the appearance that perhaps Nature is attempting, in protest, to replace these areas of lymphoid tissue which the original adenoidectomy has removed. Certainly mouth breadth, malformation of the palate, and turbinal obstruction are far more frequent causes for nasal obstruction in my experience than the adenoids. Rarely do I see adenoids so large as to cause mechanical obstruction, and it occurs to me that possibly we are a little over-zealous in the routine removal of that lymphoid tissue which may be serving a functional purpose and possibly had best be left alone unless gross infection or definite obstruction or interference of the eustachian tube ventilation is apparent.

Finally, in connection with this nasopharyngeal region I am still intrigued by what we on the eastern seaboard are told by our patients is nasal catarrh. I am interested in it because so frequently it is not the result of sinus infection. There is no posterior nasal discharge. The nasopharynx when examined in all possible ways appears to be as nearly normal as one can estimate it; yet those are the people with good turbinal ventilation and as normal a nose as one can estimate and they are the people who so bitterly complain of periodic nasal catarrh, that sensation of edema and swelling and possibly some mucoid discharge in that region. I am inclined to think that those are the patients who are best investigated, as they are being now, by the metabolic investigators, the question of thyroid secretion and excess of carbohydrate dietary measures. You are familiar with them; yet there the primary location of symptoms is in the nasopharynx and yet pathologically we see very little.

With the improved nasal pharyngoscope and a more zealous and conscientious use of the Yankauer speculum, I think that it is at least going to lead us to seek further information and a better form of examination in this unknown nasopharynx.

VERNON A. CHAPMAN, M.D., Milwaukee: I am very glad to have heard this paper by Doctor Carmody. To me it has been a long-felt need. Personally I have used the nasopharyngeal periscope routine in my examination of cases for the last twelve or fifteen years, and it is surprising to realize the information obtained. In head colds and the colds which the patient tells you he gets in the throat and then they go down into the lungs, the incipiency presents a marked inflammation of the membranes of the nasopharynx; in fact, it seems to me that all colds start that way.

I do not understand why Doctor Carmody should have so much difficulty in using the nasal pharyngoscope. There are some cases, of course, of defective septums where you cannot get through, but in those cases and in children if I use an antroscope without a sheath as a nasal pharyngoscope is used I can get through in every case. I have yet to find that I cannot pass the antroscope periscope even if I cannot pass the regular pharyngoscope.

Regarding the infections involving the nasopharynx I think they are of wide effect. I have one case of recurrent rhinitis in a young woman who resisted any therapy which yielded and did not recur after removal of greatly infected lymphoid tissue in the nasopharynx.

FRANCIS LEDERER, M.D., Chicago: I, for one, am grateful to Doctor Carmody for having presented this subject. It bears repetition every once in a while. We are grateful for the reason that this area is not the unknown so much as it is the neglected area. So often our unaccountable types of paralysis of the larynx are to be found as far as their etiological factors are concerned in the nasopharynx. Many of the so-called cases of postnasal drip, which have been tamponed to the point of economic distress, can be accounted for by close examination of these parts. It is unfortunate that because of hurried examination a nasopharyngeal examination is perhaps one of those things to which we pay least attention, but it is hoped that by Doctor Carmody's presentation it will be not the unknown but the romance will be written about this space, because it contains so much that is valuable from a clinical point of view, and when you interpret any part any closer than you do the nasopharynx you are merely skipping some definite examination that should have been carried out as a routine procedure.

I cannot believe that because a patient gags there is any reason for me to overlook this space, and certainly with the instruments that Doctor Carmody has shown and spoken about today there is no reason this space should remain the unknown. The thought that it is the cross-road is a very good one, and certainly while he is expressing all these novel ideas and resorting to the popular literature for a description, I cannot blame him at all, because constantly cases are overlooked whose etiology would be made very

clear if we carefully examined this space. While I do not contribute anything further to Doctor Carmody's already fine paper—although it does suffer from a lack of time, for which he has already apologized—at the same time I rise to thank him for bringing it before us and to thank the Academy for having such a paper presented before it.

T. E. CARMODY, M.D., in closing: I am sorry that I did not send my paper to Doctor Richards, but that is past now and I will apologize to him personally.

Doctor Richards I think misunderstood me in saying that I do not use the nasal pharyngoscope. I do use it a great deal, but there are many cases in which it is almost impossible to use it. I do not understand how Doctor Chapman can use it in almost every case, because I am unable to use it in most children. I think that I can examine a child easier with the mirror and tongue depressor than I can with the nasal pharyngoscope.

Doctor Richards spoke of using an anesthetic — a local anesthetic he means — with the Yankauer speculum. I formerly used an anesthetic, but now I simply tell the patient that I am going to hurt him a little and with adults I have no trouble. Because it is no harder to examine the naso-pharynx with the naso-pharyngoscope than it is to use a tongue depressor many of our patients because of the gaggers are difficult to examine even with a tongue depressor.

I think that Doctor Richards' suggestion of blackening the inside and putting on a distal light is very good. I have thought of putting on a distal light, but have not thought lately at least of blackening the inside.

He also speaks of the removal of the adenoids. I think that the adenoids are probably removed too much and that the tonsils are removed too early. The tonsil and the adenoid operation go together so much I am afraid that some of the pediatricians are going to catch these children just after they leave the birth canal and remove the tonsils and adenoids and there are many cases in which the adenoids are of value. There is no question that the adenoids and also the tonsils are of value as protectors, and we should not remove them too early.

Doctor Richards spoke of the interesting cases that they see on the sea coast which we probably do not see in our climate; nevertheless there are many cases of disease and infection in the nasopharynx that are overlooked.

I want to thank Doctor Lederer for bringing in the word "neglected." I think probably the title of the paper would have been better if I had said "neglected" rather than "unknown," although I think to many of us it is an unknown quantity and we do not see that upper pharynx and naso-pharynx because we do not examine carefully enough. I know that I am at fault, and I find that a great many of my friends are at fault in not making a thorough examination of that part when we do make what we think is a thorough examination of everything else. X-raying of the sinuses and so on tells us something, but an x-ray of the upper pharynx tells us practically nothing.

I think the point Doctor Lederer mentioned of tamponing is also a good one. A great many of our friends carry the word "specialists" or carry the sign "specialists" who are not specialists at all. They just simply tampon the nose. They do not make a diagnosis and they do not examine the part from which the secretion is coming, whether it is the postnasal space or where it comes from. They simply put in a pack. I have seen that happen too many times. We know well enough that a pack does very little good.

As regards Doctor Lederer's speaking of romance, I think he is probably more romantic than some of us who have reached the age that I have.

RESULTS IN THE LABYRINTH FISTULIZATION OPERATION FOR CHRONIC PROGRESSIVE DEAFNESS — REPORT OF CASES*

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IT IS THE common knowledge of all otologists that temporary improvement of the hearing in cases of otosclerosis and certain other types of chronic progressive deafness can be obtained by an operation, whereby a fistula is made into almost any part of the labyrinth. During the past twenty years considerable experimental and clinical work has been done, particularly by Holmgren, Sourdille and more recently by Lempert, seeking to establish a technic that would give a practical method of permanently improving such cases. In properly selected patients all of the earlier operators have had no trouble in obtaining an improvement of the hearing immediately on fistulizing the labyrinth, but in the great majority of instances this improvement has been maintained only a few days to several weeks because of the subsequent closure of the fistula by bony regeneration. Recently more lasting improvement of hearing has been obtained by Sourdille, according to his reports, and most encouraging reports have come from Lempert of the prolonged improvement of cases operated upon according to his technic.

It will be conceded by all having any knowledge of this subject that in suitable cases of conductive deafness a temporary improvement of hearing to a practical degree can be obtained by fistulization of the labyrinth. Moreover, it has been established that with such improvement of hearing there is also a lessening of the tinnitus so often present in such cases. Permanent improvement of the hearing and tinnitus depends almost entirely on the retention of this fistula in the labyrinth. When bony regeneration closes the fenestra the hearing either returns to its pre-operative level or becomes worse, dependent on the technic of the operative procedure.

* From the Department of Otolaryngology of University of Pennsylvania.

In the operation of such cases a favorable result may be considered obtained if there is permanent improvement of hearing to a practical level, a lessening of the tinnitus or prevention of further progression of the deafness. While all the factors necessary for obtaining favorable results are probably not as yet known at least some of them have been ascertained by experience and a discussion of these factors should be profitable.

FACTORS INFLUENCING THE RESULTS

The factors necessary for obtaining favorable results may be divided into three main headings: (A) Proper selection of the patient; (B) Proper operative technique; and (C) Proper post-operative care.

(A) Under the heading of proper selection of the patient should be considered those factors that appear to make the patient a suitable one for operation. These factors concern particularly (1) the degree of deafness, (2) the status of the bone conduction, (3) the adequacy of the vestibular function and (4) the state of the drum, middle ear and canal wall.

1. Degree of Deafness: The amount and type of deafness become important considerations in the selection of patients for the labyrinth fistulization operation. In order that the hearing may be improved to a practical level it is necessary that the initial deafness be not greater than 60 or 65 per cent (estimated by multiplying the average audiometer readings at 512, 1024, and 2048 frequencies by 0.8). However, if the other factors remain favorable a degree of deafness of 70 or 80 per cent need not contra-indicate the operation, but an improvement of 30 to 35 decibels at the critical frequencies would still not bring the hearing to a practical level; although such improvement may be beneficial to the patient particularly if the tinnitus is lessened. The complete loss of hearing to the highest frequencies such as 5792, 8192 and those higher is not an unfavorable prognostic sign if the hearing for the speech frequencies remain reasonably good. It may be questioned if patients with less than 35 or 40 per cent loss of hearing should be operated because such patients still have some serviceable hearing which may be diminished if the operation is not completely successful. It seems perfectly justifiable to operate patients with this degree of hearing or even less than 30 per cent loss if it can be determined that such deafness is rapidly progressive, or is accompanied by disagreeable tinnitus, or in cases where the patient's

business or profession is such that even the milder degrees of deafness become a handicap. In the consideration of patients for operation it must be remembered that the operative procedure seeks to lessen the tinnitus and prevent further progression of the deafness, as well as to improve the hearing.

2. Status of the Bone Conduction: From the experience gained in the observation of operated cases it appears of extreme importance that the bone conduction remain good if good results are to be expected. The factors concerned in bone conduction are intricate and incompletely understood so that it is difficult to give any definite standards that will determine whether a particular patient is or is not suitable for operation as far as this factor is concerned. If a bone conduction audiometer receiver is available where a standard pressure can be maintained against the bone to be tested and a suitable masker used, then this method becomes of value in the estimation of the bone conduction and definite figures can be obtained for each frequency.

The determination of the bone conduction by means of the tuning forks is a simple but apparently effective way of estimating the suitability of the patient for operation as far as this factor is concerned. When the tuning forks show a lengthened bone conduction and a markedly negative Rinne test and a Weber test giving lateralization to the deafer ear it is good evidence that this factor of bone conduction is favorable. When the bone conduction is found to be less than the air conduction with lateralization to the better ear, such a case should, as a rule be considered unfavorable for the fistulization operation, although it is quite possible that such a patient may still be improved by operation because the estimation of the bone conduction may have been at fault due to unknown factors that are known to interfere at times with the conduction of sound by bone.

3. Adequacy of the Vestibular Function: Normally functioning semi-circular canals appear to be an important prerequisite for good results. It is quite possible for the vestibular mechanism to be dead and the chochlear function remain unaffected. In such cases on fistulizing the labyrinth the dizziness, nausea and nystagmus observed when an actively functioning labyrinth is fistulized, will be absent. This was exemplified in one of my cases (Case No. 7) reported a few months ago. On opening the external semi-circular canal no dizziness or nausea resulted and even after the membranous canal had been opened no dizziness or nystagmus fol-

lowed. In this case it was also observed that there was not the immediate improvement of hearing when the membranous labyrinth was exposed as is expected in the suitable cases. Similar experiences have not been reported and no definite statement regarding this point should be made from the observation of one case, but it seems logical and probable that improvement of hearing will not be obtained by the fistulization of a dead labyrinth. It is important therefore, before considering operation on a patient, to test the vestibular function by means of the Barany or Caloric tests. I believe those patients with weakened or absent vestibular function should be considered as unsuitable for operation.

4. State of the Canal Wall, Drum and Middle Ear: A healthy condition of the skin surface lining the canal wall, particularly the upper and posterior surfaces, seems essential. Any eczema or dermatitis of this area may cause post-operative inflammation or irritation that would impair the results or may possibly set up a labyrinthitis. An exostosis of the deeper canal wall may make it difficult or impossible satisfactorily to dissect free the cutaneous lining of the bony canal wall without lacerating it. It has been supposed that a perfectly normal tympanic membrane without perforations is essential for good results. It is quite likely that such a condition is necessary for the best results but a drum that is thickened, retracted and scarred need not contraindicate operation if the other factors remain suitable. Three of my cases in which at least fair results were obtained have had thickened and scarred drums with the history of having had suppurative otitis media at one time. It is also questionable if a patient with a perforated drum is still suitable for operation. The state of the middle ear may have some bearing on the results to be obtained by operation. It is supposed that the best results will be obtained in those typical cases of otosclerosis in which the drum is entirely normal and there is fixation of the stapes. It seems very likely true that the best results will be obtained in such cases, but I believe further experience will indicate that at least good results may be expected when the drum is not entirely normal and when there is not marked fixation of the stapes. The presence of an acute severe suppurative otitis media at one time may have caused some destruction of middle ear structures, fibrosis, adhesions, etc., which may interfere to some extent with good results of the operation. More experience is necessary to determine if and how much such factors influence the results in the fistulization operation.

(B) Proper Operative Technic: While the proper selection of the patient is of prime importance the results of the operation would be poor or nil without close adherence to an exacting operative technic. The basis of this proper technic is an anatomical knowledge of the parts involved, skillfulness and care in the handling of the most delicate structures, patience and physical endurance. To obtain results according to Lempert's technic these requirements are strictly necessary. This procedure is difficult and without doubt there will be attempts to modify and simplify it, but until such modifications have proven themselves, the Lempert technic will to my mind remain the one of choice among those that have been devised thus far for the surgical treatment of chronic progressive deafness.

There are certain particular points of this technic that are of special importance and should be given emphasis. From the beginning of the operation and throughout its course the greatest possible care must be exercised for the preservation of the cutaneous membrane lining the posterior and superior walls of the external auditory canal. In the removal of the bony canal wall and sulcus tympanicus, this membrane will be lacerated and destroyed unless extreme care is taken. Another delicate part of the technic is the exposure of the malleus. In this procedure it is easily possible to dislocate the incus requiring its removal or to lacerate the tympanic membrane. The removal of the sulcus tympanicus, particularly in its upper portion is an intricate part of the technic and requires great care and patience to avoid lacerating the drum. Finally the success of the operation depends on the proper technic in creating the fistula in the external semicircular canal and the fitting of the tympanomeatal cutaneous membrane over this fistula. Incomplete exposure of the membranous labyrinth may result in prompt closure of the fistula by bony regeneration, while too much exposure may result in a perforation of the membranous labyrinth. The success of the operation depends almost entirely on the proper handling of this part of the operation, although the previous steps are of importance, if the greatest possible amount of hearing is to be gained. When the fistula is completed there is an immediate improvement of hearing, but for this improvement to be maintained at its highest level it is necessary that the previous stages of the operation be completed accurately. For instance if the tympanomeatal cutaneous membrane be lacerated, there will be no membrane available to cover the fistula, unless a new flap is dissected

from the lower or anterior canal wall and then it will have no connection with the drum. If the drum itself is lacerated the healing process may cause a retraction that will impair its efficiency. If the incus is dislocated the ossicular chain is broken and any sound impulses that are being carried through that channel are lost. When such accidents occur, however, the operation need not be considered a complete failure for improvement of hearing will still be present if the fistula remains open. This is illustrated in my first case operated 15½ months ago. During the early stages of the operation the cutaneous lining of the external auditory canal was badly lacerated so that it was impossible to use it to cover the fistula in the external semicircular canal. A skin flap was therefore dissected from the anterior canal wall and stretched across the canal lumen and placed so as to cover the incus, the posterior edge of the tympanic membrane and the fistula of the external semicircular canal. In the healing process this resulted in a narrowing of the canal lumen to about one-half its normal size with a band of tissue stretched across the lumen from the anterior to the posterior walls, with the drum visible below this band and the area of the antrum visible above this band. I consider this patient one of my best results as an audiogram on August 29, 1939 — fourteen months after operation — shows an improvement of hearing from its preoperative level of 20 decibels for the 512 frequency, 30 decibels for the 1024 frequency, 35 decibels for the 1448 frequency and 25 decibels for the 2048 frequency or an improvement of 21.5 per cent. The fistula reaction remains active. In another patient both the cutaneous lining of the canal wall and the drum were lacerated but the remnants of the membrane were approximated as closely as possible and a section of the torn cutaneous membrane placed carefully over the attic cavity, the incus and the fistulized external semicircular canal. These torn skin sections were kept in place by small pieces of gauze mesh. This patient's ear healed so well that the drum barely shows a scar of the laceration and the skin has completely lined the excavated mastoid cavity and antrum. An audiogram on August 25, 1939, seven and one-half months after operation, showed an improvement of hearing over the preoperative level of 15 decibels for the 512 frequency, 30 decibels for the 1024 frequency, 35 decibels for the 1448 frequency and 35 decibels for the 2048 frequency or an improvement of 20.6 per cent. It seems quite probable that greater improvement of the hearing in those two patients may have been obtained if these accidents in the

operative technic had not occurred but they illustrate that such an apparently serious fault may still be consistent with at least a fairly good result.

It has been asked in what way the removal of the head of the malleus contributes to the results obtained. The reason for its removal appears to be that greater mobility of the drum is gained and makes it possible to stretch the tympanomeatal cutaneous membrane to such an extent that the thinnest part of this skin membrane and probably frequently Shrapnell's membrane itself, can be placed directly over the fistulized semicircular canal. It also apparently facilitates the sealing off of the epitympanic space. Whether or not the removal of the malleus head is necessary for good results in all cases is a matter for further experimental study. In three of my more recent cases I have not removed the head of the malleus and the immediate results have been good. It is too soon after operation, however, to be able to form any definite opinion on this particular point but it is a matter I shall discuss later when time will have permitted further observation and study.

In general, as regards the value of the operative technic it can be said that good results depend very largely on the proper method of fistulizing the labyrinth and that the other details of the operative procedure will probably add to the results obtained in proportion to the accuracy and completeness of the various steps. However, it can also be said that minor accidents in the technic may still allow of an improvement of hearing that may be considered good.

(C) Proper Postoperative Care: This is the least important of the factors that may influence the result of the fistulization operation, although carelessness or negligence in the post-operative care may lead to failure in an otherwise successful case. The chief things to consider under this heading are (1) maintenance of asepsis (2) treatment of excessive granulations and (3) general care and advice.

(1) Maintenance of Asepsis: It is just as important to prevent bacterial contamination of the operative site after operation as it is to insure an aseptic field before operation. This may not be easy to do as the ear requires frequent cleansing and dressing. Furthermore, carelessness on the part of the patient may allow the ear to become contaminated. Following the operation there is considerable thick exudative moisture in the operated area and covering the canal walls and drum. It is advisable that this exudate

be allowed to drain away and therefore it is necessary that the patient change the sterile cotton or gauze dressing over the ear frequently, often two or three times a day. The patient must be warned to use care in doing this and to use only sterile materials. One of my patients developed a middle ear abscess, in the operated ear, six weeks after operation. The drum perforated and drained pus for about a month. This caused an immediate decrease in the hearing but no systemic effects and no symptoms of labyrinthine stimulation. Before the abscess developed the hearing had improved 10 decibels for the 512 frequency, 30 decibels for the 1024 frequency and 30 decibels for the 2048 frequency. During the course of the ear infection the hearing was reduced from this level and for the past five months has remained at practically the same level, the last recording on September 1, 1939, being 38.7 per cent loss, almost exactly the reading before operation. As the fistula has remained open as indicated by an active fistula reaction, I have attributed the reduction of the hearing to changes set up by the ear infection.

(2) Treatment of Granulations: Exuberant granulations in the operative site are common and require burning with trichloracetic acid (25 per cent) or strong silver nitrate solution. Application of such agents also stimulate epidermization of the cavity. There will be an exudative discharge and formation of granulations for two to four months after operation. This necessitates frequent attention by the surgeon as well as care by the patient during this time.

(3) General Care and Advice: After the patient is discharged from the hospital usually the post-operative dizziness has subsided sufficiently as to require no particular care. At times, however, the dizziness will persist for several weeks particularly on stooping or turning movements. The patient must be cautioned against over-stimulation of the labyrinth by exertion. On the other hand, he should be encouraged to maintain some activity in order to accustom himself to the changes in the equilibrium mechanism. The patient is apt to be apprehensive of this frequent vertigo but apparently there is gradual adjustment of this reaction, and later does not interfere with normal activity, although quick turning, cold air in the ear, etc., may cause more dizziness than had been present before operation.

The patient may require some treatment and advice during the convalescence because of the changeable character of the hearing.

After the gauze packing is removed from the ear at the end of seven or eight days, considerable improvement of hearing is usually noticeable. The hearing may be greater at this time than later when the tissues have healed but at times the trauma to the tympanic membrane and other tissues may cause such a swelling and edema with perhaps some hemorrhage into the middle ear cavity that improvement of hearing is delayed until this reaction has subsided. Furthermore, a serous labyrinthitis may have developed from manipulation of the membranous labyrinth and this will delay improvement of the hearing, as well as increase the vertigo until this labyrinthitis subsides. During the weeks following operation the hearing will vary somewhat according to the amount of moisture present and how well it is drained off. Frequently there is considerable thick exudate that covers the drum and fistula area preventing the transmission of sound impulses. Frequent removal of the secretions is advisable to prevent this. If the patient develops a nasal or sinus infection during the convalescence, it is apt to reduce the hearing due to inflammatory reaction in the Eustachian tube. This is similar to the impaired hearing accompanying many colds in anyone and the hearing improves again as the nasal and tubal inflammation subside. I have noticed this on several patients and it is cause for apprehension on their part as to the effects on their hearing. These patients are for the most part of a nervous and emotional temperament and require careful handling and frequent encouragement.

The factors which have been discussed have a variable effect on the results obtained with the labyrinth fistulization operation. It is again emphasized that accidents during the operative technic may perhaps lessen the final result obtained, but may not necessarily cause a failure in the particular case operated. Permanent improvement of the hearing depends on the maintenance of the fistula in the labyrinth, and on the accuracy with which the other details of the operation are conducted depends to a large extent the amount of hearing that will be recovered.

REPORT OF CASES

At this point I should like to give a brief follow-up report on those cases which were reported seven months ago and which will be published within a short time. It is still a questionable point how long a time must elapse before it can be said that the danger of closure of the fistula by bony regeneration is past. It is only by close follow-up observation of operated cases that this point can

be settled. The following is a brief outline of the progress of the eight cases reported 7 months ago.

Case No. 1 (E. H.). A man, aged 35, was operated on June 29, 1938, on the right ear. A pre-operative audiogram showed (by air conduction) a 54.7 per cent loss of hearing in the right ear and 42.7 per cent loss in the left ear. Six months after operation an audiogram showed 35.5 per cent loss in the right ear, two months later 34.4 per cent loss and on August 29, 1939, 33.3 per cent loss. At the end of 14 months following operation therefore the improvement of hearing is 21.4 per cent greater than before operation (a gain of 20 decibels for the 512 frequency, 35 decibels for the 1024 frequency and 30 decibels for the 1448 frequency and 25 decibels for the 2048 frequency). Audiograms of the unoperated ear during this time have shown a slight increase of the deafness. The fistula reaction 14 months after the operation is very active.

Case No. 2 (S. H.). A man, aged 43, had the fistulization operation on July 13, 1938. An audiogram on July 12, 1938, showed 40 per cent loss of hearing in the right ear and 38.1 per cent loss in the left. His bone conduction was not good and he was operated because the tinnitus was very distressing and it was thought there was some possibility of improving it. The operation was a failure as the membranous labyrinth was perforated, resulting in the loss of the vestibular function. The hearing after operation was moderately reduced and has not improved to date and the tinnitus has not become less. There is, however, some hearing still present in the operated ear and the bone conduction has not changed.

Case No. 3 (C. P.). A girl, aged 19, was operated on July 20, 1938. There was immediate improvement of the hearing, but it began to fade soon and at the end of three weeks from the operation, the hearing had diminished below what it was before the operation and the fistula test was negative. On December 8, 1938, the external semicircular canal was again fistulized with again good immediate results. Within a few weeks, however, the hearing began to diminish with lessening fistula reaction and on August 29, 1939, an audiogram showed 57.3 per cent loss of hearing, a decrease of 16.5 per cent from the preoperative hearing. There is now no fistula reaction present and the tinnitus which had been improved at first is present as before.

Case No. 4 (E. W.). A girl, aged 21, received operation on the right ear on August 29, 1938. Preoperative audiograms showed

a hearing loss of 35.5 per cent in the right ear and 27.7 per cent in the left ear. Immediately following the operation the hearing was much improved but within two weeks it had begun to diminish and at the end of one month from the operation the hearing was slightly worse than before operation with return of the tinnitus. Her labyrinth was re-fistulized on January 5, 1939, with again improvement of hearing. During the subsequent few months audiograms showed a maintenance of improvement but the improvement has not been great. On August 29, 1939, just one year after operation, there was 30.7 per cent loss of hearing — an improvement of 5 per cent over the pre-operative hearing. The fistula reaction is still present, but not as active as during the first month following operation. There has been no change in it, however, during the past three months and no change in the hearing.

Case No. 5 (N. G.). A woman of 44 had the right external semicircular canal fistulized on September 30, 1938. She had a successful radical mastoid operation performed on the ear some years before. The present operation consisted in elevating the thin skin overlying the antrum and aditus region, fistulizing the external semicircular canal and replacing the skin over the fistula. A preoperative audiogram showed a hearing loss of 25.9 per cent. During the four weeks following the operation the hearing was worse than before operation coincident with severe dizziness, nausea and vomiting which was attributed to a serous labyrinthitis set up by the operation. As this subsided during the second month the hearing improved but did not return quite to the preoperative level. An audiogram on August 22, 1939 (about eleven months after operation) showed a hearing loss of 34.7 per cent, a reduction of 9 per cent over the preoperative level. The fistula reaction remained very active for the three months after operation, but during the next month became gradually less active. For the past five months there has been no change in its reaction, but it is not nearly as active as during the earlier months. A peculiar feature of the case is the fact that at times there is considerable improvement of the hearing for periods of a few minutes to a day or two without any apparent reason.

Case No. 6 (N. C.). A woman of 38 had the fistulization operation on October 12, 1938. Preoperative audiograms showed a loss of hearing in the right ear of 61.6 per cent and in the left ear 48.8 per cent. She gained considerable immediate improvement but this became worse during the first three weeks associated with severe

dizziness and nausea. When this subsided there was marked improvement and at the end of a month the hearing was better by 30 per cent. Three months after operation the hearing was improved by 25.3 per cent and the last audiogram on September 18, 1939, showed 41.3 per cent loss or an improvement of 20.3 per cent over the preoperative level (a gain of 25 decibels for the 512 frequency, 30 decibels for the 1024 frequency, 20 decibels for the 1448 frequency and 20 decibels for the 2048 frequency). It has now been almost exactly a year since the operation and the fistula test gives an active response. The hearing is not as good as at the end of the first month following operation, but the practical improvement is great and has been extremely pleasing to the patient.

Case No. 7 (L. H.). A boy, aged 14, received his operation on the left ear on October 19, 1938. Preoperative audiograms showed 46.9 per cent loss of hearing in the right ear and 53.3 per cent loss in the left ear. There had been a history of ear abscesses earlier in life and the drums showed thickening, retraction and scarring. The bone conduction was somewhat decreased with lateralization of tone to the better ear. When the external semicircular canal was fistulized there was not the usual dizziness and improvement of hearing noticeable in the other cases. During the few weeks following operation audiograms showed about 20 per cent loss of hearing and to date it has not improved. The patient is unaware, however, of the decrease of hearing because his preoperative hearing was so little that no difference is noticeable to him.

This case illustrates well the unsuitability of the patient for operation because of the decreased bone conduction and dead labyrinthine function.

Case No. 8 (F. B.). A woman of 40 was operated on January 11, 1939. Preoperative audiograms showed a hearing loss in the right ear of 61.9 per cent and in the left ear of 40.3 per cent. At the end of six weeks following operation the hearing of the operated ear (right) had improved to 25.3 per cent loss (an improvement of 36.6 per cent), (a gain of 40 decibels for the 512 frequency, 40 decibels for the 1024 frequency, 60 decibels for the 1448 frequency and 45 decibels for the 2048 frequency). During the next five months there was a partial loss of the improvement until on July 28, 1939 (6½ months after operation), the hearing loss was 40 per cent, an improvement of 21.9 per cent over the preoperative level. Although the greatest improvement of hearing gained by this operation has not been maintained, there is still

considerable improvement which has been extremely beneficial to the patient. The fistula at present is active but not as active as during the first 2 months after operation. It must be assumed that there has been some regeneration of bone which has partly closed the fistula. As there has been no further hearing loss in the last 2 months it seems that this improvement will be maintained.

In a short time, I shall make a preliminary report of 10 additional cases operated within the last few months. The early results in most of these new cases have been gratifying but it is too early to be sure that the improvement of hearing thus far obtained will remain.

An analysis of the eight cases described above will reveal results of a variable character. Three of the cases have gained decided improvement of hearing, the first having retained an improvement of 21.4 per cent after 14 months, the second, an improvement of 20.3 per cent one year after operation and the third, an improvement of 21.9 per cent eight months after operation. Two cases showed prompt closure of the fistula by bony regeneration. On reopening these external semicircular canals one of these cases has again shown closure of the fistula with a loss of 16.5 per cent over the preoperative hearing. The second of the re-operated cases has retained a fairly active fistula and an improvement of 5 per cent in the hearing at the end of nine months from operation. In two of the cases the hearing has definitely been made worse, due to their being unsuitable for the operation, both having had decreased bone conduction and one having had a dead labyrinth. The remaining case, the one in which fistulization was attempted in a previously radically operated mastoid, has also shown a slight loss of hearing over the preoperative level.

The results obtained in these few cases emphasize the importance of those factors that have an influence on the success of the operation. The proper selection of the patient remains the prime prerequisite while the proper operative technic and post-operative care are of somewhat less importance, but still extremely necessary, if the greatest amount of hearing is to be gained. Four successful cases out of eight will not be considered as very good results, but if it can be shown that these cases have gained a practical improvement of hearing, or a reduction of the tinnitus and have retained the fistula beyond the time that bony regeneration should have occurred then the operation can be considered as demonstrating the possibility of permanently improving the hearing. As the

standards for selection of the patient become understood and the operative technic be developed and improved, it seems reasonable to assume that a high percentage of patients will obtain improvement of the hearing by such surgical procedures.

DISCUSSION

SAMUEL J. KOPETZKY, M.D., New York: I depart from the custom of confining my remarks to comments upon what has been presented by Doctor Campbell. I do so because his presentation is a record of operations performed and results obtained. It stands as a record of competent work and reliable observations. I can, from my own experience, substantiate his exceptions to standardized procedure. In his report anent the "trial and error" method of doing something for deafened patients, he makes no final evaluation because of his limited period of observation, but he promises us further reports on his cases.

Like Campbell, for many years the problems which concerned the deafened have interested me, and after I had studied the prospects which surgical therapy promised, I drew the attention of American otology in May, 1938, to the probabilities of achieving beneficial results in cases of deafness by surgical therapy; and *at that time,* particularly and specifically, I directed attention to the operation perfected by Lempert, and I advocated that it be tried — leaving to further study its eventual place in our therapeutic armamentarium.

I am, however, but little interested in a particular surgical technique, because in the long run, this question will settle itself by the "trial and error" method. But the multiplicity of the problems presented by fistulization for the relief of the deafened is a challenge to me and to other workers in this field, which cannot be left unanswered.

One may divide all reports on surgical therapy for the deafened, into three phases. The first, the occasional, casual and cautious attempts which are scattered in the literature. The second is the empirical application of surgical measures. Here I place Sourdille's operative reports, and the reports found in our own literature. The third is the study of the problems which this surgery brings in its train, and to the solution of which only the very beginnings have been made. In fact, most of the problems have not, as yet, been clearly defined or even enunciated. A commencement has been made by Holmgren, who seeks the establishment of fundamental factors upon which to predicate a rational surgical therapy. His studies of various means to keep the labyrinthine fistula open, and Nager's histological studies are the first efforts to take fistulization out of the realm of empiricism and into one of rationalism based upon adequately observed factual data. Canfield, too, is engaged in finding some rational basis for this type of surgery. To this date, that is all that otological literature reveals along this line. What Canfield promises to undertake should clarify many perplexing questions, which reports on operated cases cannot settle.

My own work is not yet ready for final report. I will, however, take this opportunity to present some "sample cases" from my material, to draw at-

tention to some characteristic features — some of which definitely substantiate Campbell's presentation today, and a few others which present their own arguments in contrast to his. I shall let you make your own deductions.

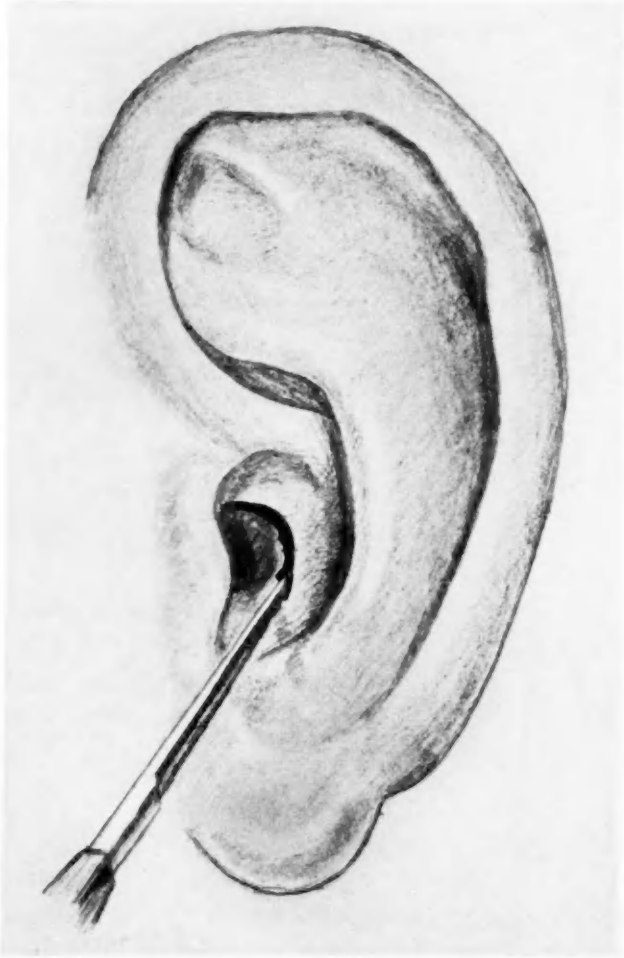


FIG. 1 — (Kopetzky) — Incision of superoposterior edge of external auditory canal with concha, to free flap.

If the efforts to acquire the necessary technical dexterity to perform this type of surgery leads you to the dissecting room, I hope my contribution will drive you again to the physiological laboratory, because the whole question of "how we hear," what structures come into play "while hearing" and "while listening" is in need of clarification. Theories of hearing, based

upon premises which in themselves are equivocal, need re-examination. They are largely built upon unproved foundations, and the very light which is being shed by the results obtained by our empiric operative procedures may perhaps lead to new conceptions in the fundamentals of the physiology of hearing.

In confirmation to Doctor Campbell's exceptions to standardized procedures, I state that the endaural approach must not be considered the essential approach. There are no objections to its employment by those who desire to use it to expose the temporal bone, but it should be emphasized that all steps in the technique of labyrinthine fenestration can be done

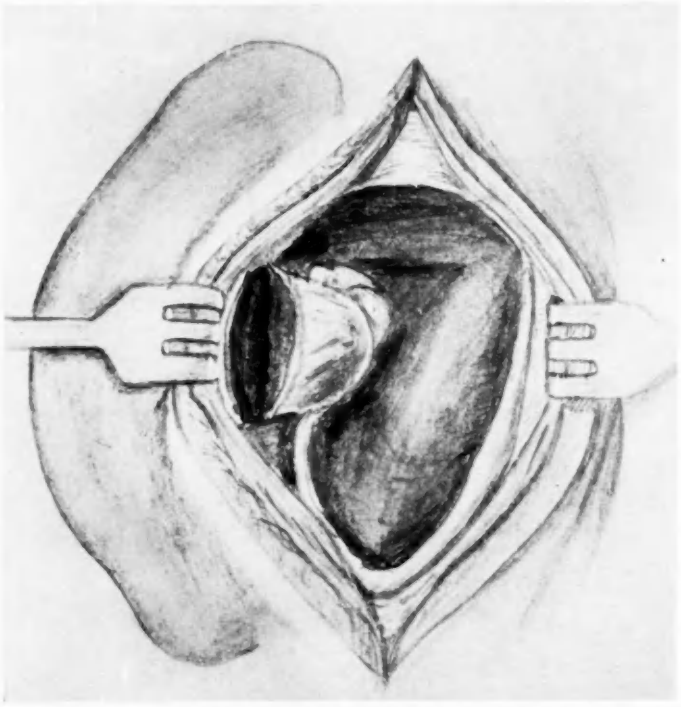


FIG. 2 — (Kopetzky) — Exposure obtained by freeing concha, turning it toward the face, showing cut surface of external auditory canal wall. Also remaining part of membranous wall, bony wall removed. Epitympanic space exposed. Partly diagramatic.

equally well through the usual, the open postauricular route; and the same direct vision, the same inter-relationship of parts is equally well obtainable. For those of us who have resident and staff assistants at operations, who must be trained in otolaryngology, the open, post-auricular approach offers obvious advantages. (See Fig. 1 and Fig. 2.)

In using the postauricular approach, this figure shows the incision to be made as the first step in the technique. Fig. 2, which follows, shows the exposure obtained when the concha is retracted toward the face, and the mastoidal, and epitympanic areas have been exposed.

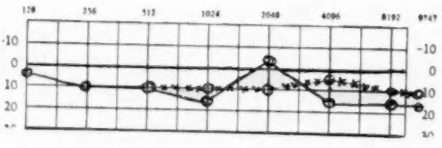
The kind of membrane which covers the fenestration is not the factor responsible for the permanence of a labyrinthine fistula. This Canfield has shown experimentally; and my own observations on the living patient, as well as Campbell's, substantiate his findings. Nor is the following of an exactly outlined, definite technique the *"sine qua non"* which determines success or failure. Campbell implies this too, and I agree with him.

Today I show sample cases. Only the pertinent factor in each is stressed. The complete case report will be published when more time for observation has elapsed. I therefore present only such data as will serve to elucidate the observations of Campbell and myself.

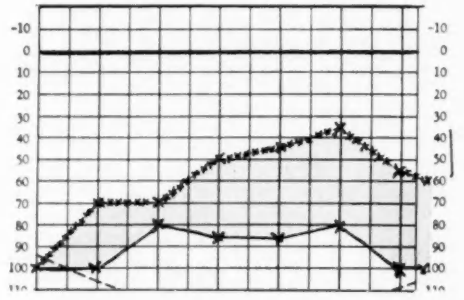
MRS. S. W. age - 22

LEFT EAR - unoperated side.
Shaded section denotes decibel gain since operation. Chronic discharging ear. Radical mastoidectomy on right ear for cholesteatoma, Aug. 25, 1937. Fistula into horizontal semicircular canal found at operation, lined with cholesteatoma matrix, which was left in situ. Usual operative procedures carried out.
Record from Aug. 16, 1937 to Nov. 4, 1937.

Starred line indicates postoperative curve.



RIGHT EAR - operated side.



RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Aug., 1937	65%	7%
Nov., 1937	44%	8%

FIG. 3 — (Kopetzky)

This is a case of a spontaneous fistula covered with cholesteatomatous matrix. The cholesteatome was removed surgically. There is now an epidermatized radical cavity. The hearing acuity is definitely improved, after operation. (See Figs. 3 and 4.)

Fig. 3 is a self-explanatory contracted chart of the case. I draw attention to the excellent hearing acuity in the normal, *unoperated* ear, in contrast to what I show later on in other types of cases.

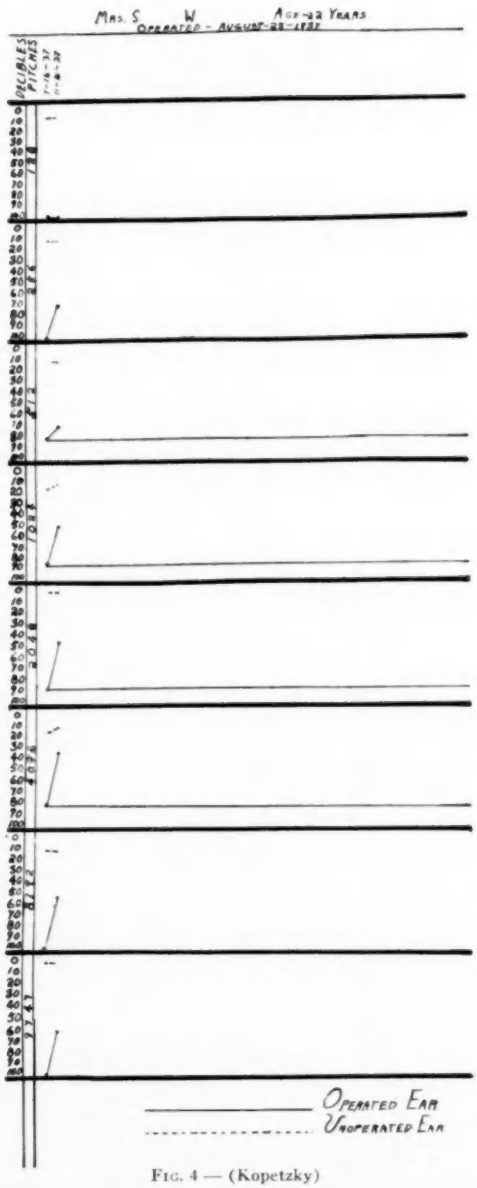
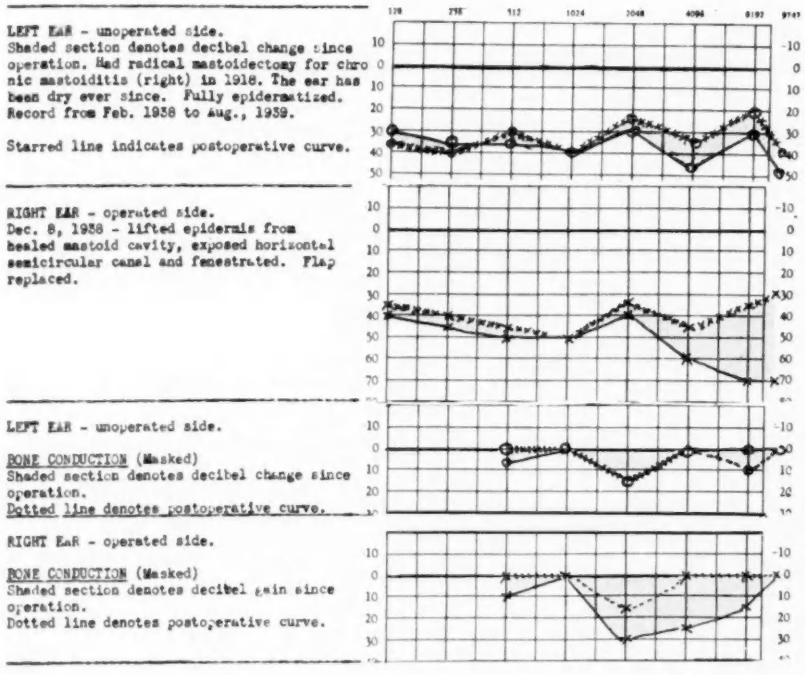


FIG. 4 — (Kopetzky)

Fig. 4, and others which will follow, is a "breakdown" of the several audiometric recordings, so that the record for all tones over the period of observation may be studied separately. Thus it is possible to record many successive audiometric tests and analyze the trend of the curve for each note.

The following is a case in which a healed radical mastoid cavity was taken after 20 years, and the horizontal semicircular canal fistularized. Fig. 5 is a contracted chart of this case, which is self-explanatory.


MR. I. G. Age - 32
LEFT EAR - unoperated side.
Shaded section denotes decibel change since operation. Had radical mastoidectomy for chronic mastoiditis (right) in 1918. The ear has been dry ever since. Fully epidermatized. Record from Feb. 1938 to Aug., 1939.
Starred line indicates postoperative curve.
RIGHT EAR - operated side.
Dec. 8, 1938 - lifted epidermis from healed mastoid cavity, exposed horizontal semicircular canal and fenestrated. Flap replaced.
LEFT EAR - unoperated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel change since operation.
Dotted line denotes postoperative curve.
RIGHT EAR - operated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel gain since operation.
Dotted line denotes postoperative curve.

RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Feb., 1938	37%	28%
Aug., 1939	34%	25%

FIG. 5 — (Kopetzky)

Fig. 6 is a chart of the hearing acuity for all notes on the Western Electric 6-A Audiometer, from December 7, 1938, to August 18, 1939. There is an improvement for all tones. There is also an improvement for all tones in the unoperated ear. The fistula remained open and active until May — a period of five months. The hearing remains at this level *even though no fistula test reaction can now be produced.*

This case is shown to demonstrate that the presence of tympanic structures is not essential to a successful result from a fistulization operation. The opposite ear also improved. The fistula closed after an intercurrent upper respiratory infection. The hearing at this time still remains improved.

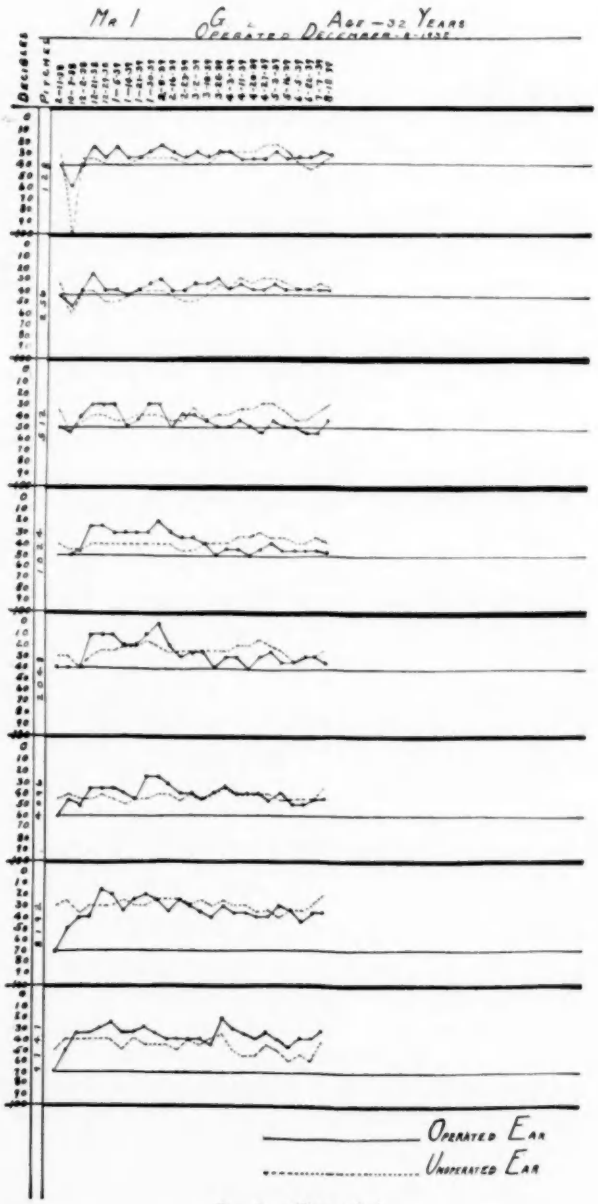
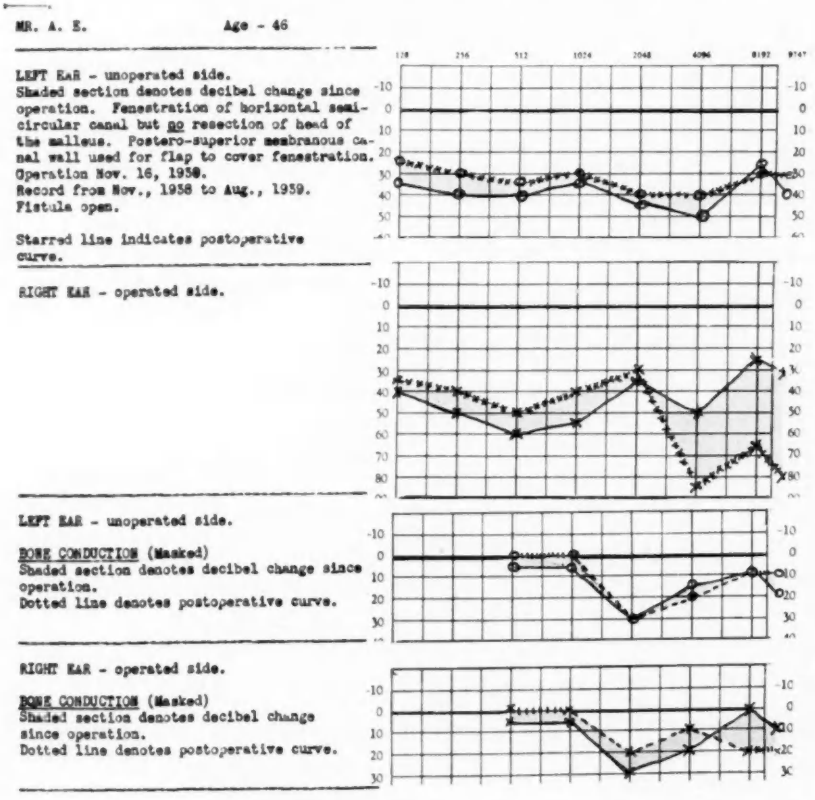


FIG. 6 — (Kopetzky)

In these two cases, one with a spontaneous fistula, and one with a surgically made fistula, radical mastoidectomies had been done. Note the improvement of hearing in the unoperated ear, for most tones.

A case is shown in Fig. 7 wherein the middle ear and the epitympanic space have been left *undisturbed*. The rationale for interfering with these


MR. A. E. Age - 46
LEFT EAR - unoperated side.
Shaded section denotes decibel change since operation. Fenestration of horizontal semicircular canal but no resection of head of the malleus. Postero-superior membranous canal wall used for flap to cover fenestration.
Operation Nov. 16, 1958.
Record from Nov., 1958 to Aug., 1959.
Fistula open.
Starred line indicates postoperative curve.
RIGHT EAR - operated side.
LEFT EAR - unoperated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel change since operation.
Dotted line denotes postoperative curve.
RIGHT EAR - operated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel change since operation.
Dotted line denotes postoperative curve.

RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Nov., 1958	40%	32%
Aug., 1959	32%	28%

FIG. 7 — (Kopetzky)

structures has hitherto never been satisfactorily clarified. To test the advantage or the disadvantage of leaving the epitympanic space undisturbed, I first exenterated the mastoid process, extended the procedure to outline sharply the horizontal semicircular canal — which was fenestrated; and then the membranous posterior meatal wall in its entirety was adapted to cover the fenestrum. The hearing acuity is shown on Fig. 7 and Fig. 8.

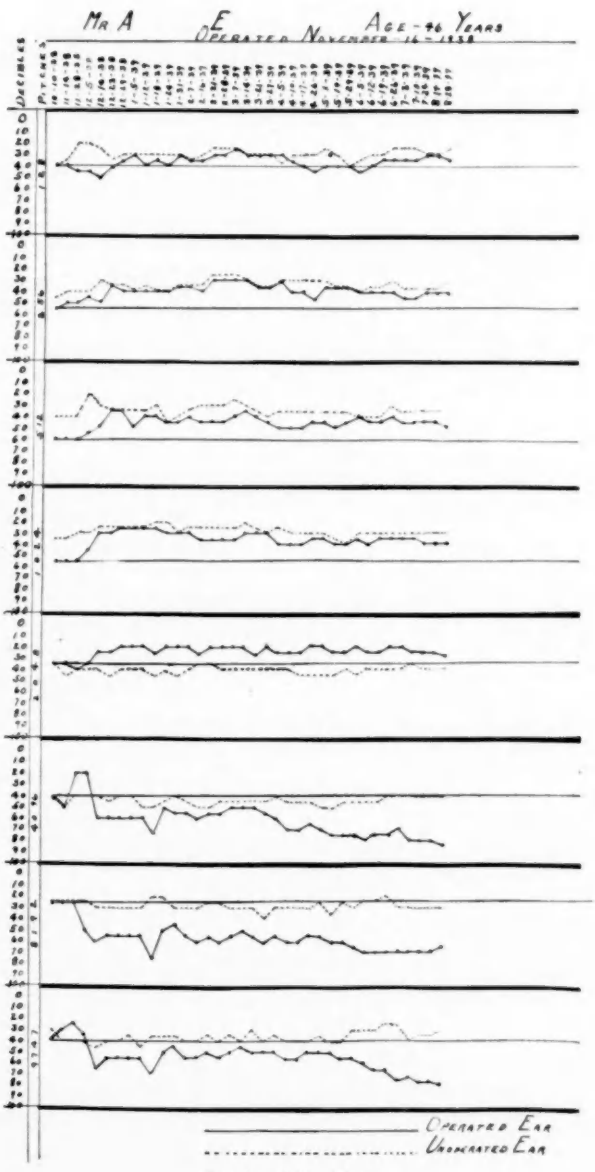
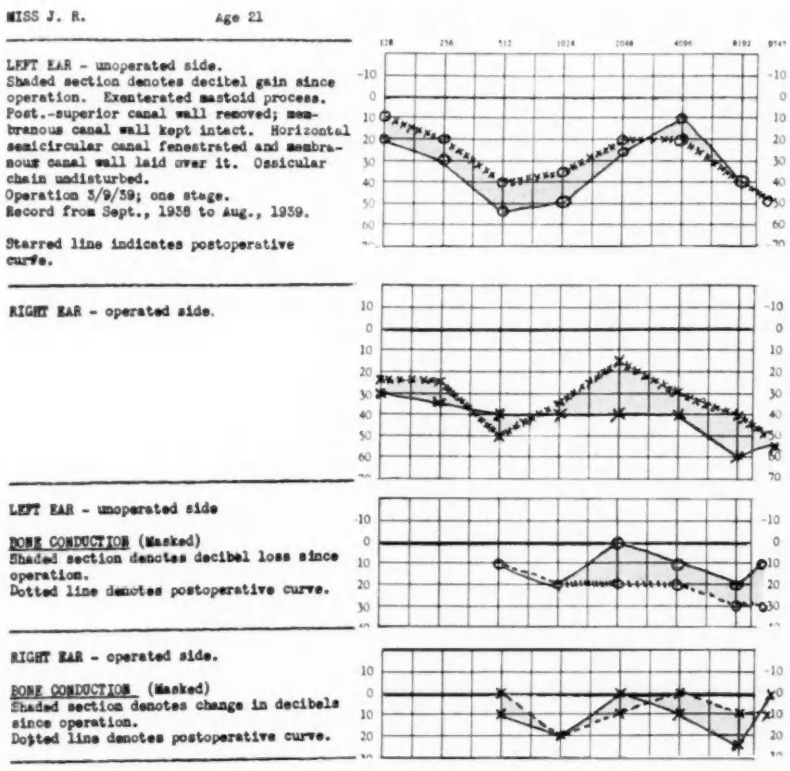


Fig. 8 — (Kopetzky)

Hearing for conversational tones has improved 8% in the right ear and 4% in the left. From all standpoints these charts *are unimpressive*, and one could not call this case a success, despite the fact that the fistula is still active. Yet this small gain in hearing acuity satisfies the patient, and he is contented. Note that the hearing in the *unoperated* ear has also improved since operation.


MISS J. R. Age 21
LEFT EAR - unoperated side.
Shaded section denotes decibel gain since operation. Exenterated mastoid process. Post.-superior canal wall removed; membranous canal wall kept intact. Horizontal semicircular canal fenestrated and membranous canal wall laid over it. Ossicular chain undisturbed.
Operation 3/9/39; one stage.
Record from Sept., 1938 to Aug., 1939.
Starred line indicates postoperative curve.
RIGHT EAR - operated side.
LEFT EAR - unoperated side
BONE CONDUCTION (Masked)
Shaded section denotes decibel loss since operation.
Dotted line denotes postoperative curve.
RIGHT EAR - operated side.
BONE CONDUCTION (Masked)
Shaded section denotes change in decibels since operation.
Dotted line denotes postoperative curve.

RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Sept., 1938	32%	34%
Aug., 1939	28%	24%

FIG. 9 — (Kopetzky)

At this point permit me to stress that while there is no better criterion for estimating hearing acuity than the audiometer, both for accurate recordings and for standards of comparison, it is apparent to anyone who operates on these cases that much more is accomplished than what the audiometer records. I shall revert to this again later.

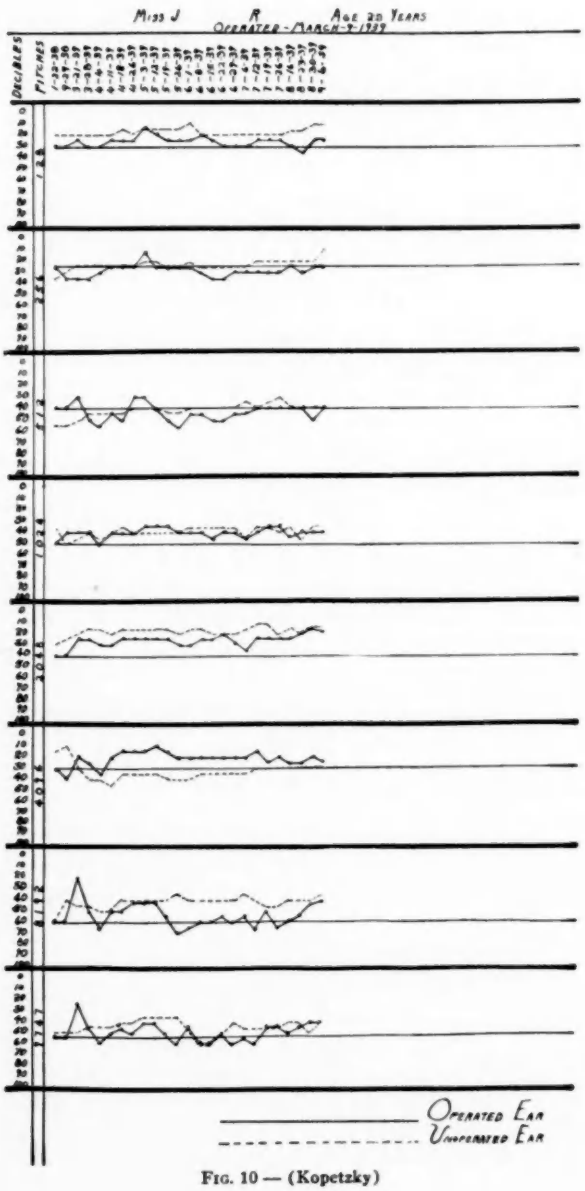


Fig. 10 — (Kopetzky)

In the case sketched in Fig. 9, the tympanic and the epitympanic spaces were also left *untouched*. The horizontal semicircular canal was fenestrated and covered by the flap from the posterior membranous canal. The chart of the hearing in this case definitely is *not impressive*. In the conversational range of the 512, 1024 and 2048 octaves, there is no marked improvement, even though the fenestration remains open. There is some degree of improvement in hearing, found in the operated ear as well as in the unoperated ear. The parallelism in the curves in both ears is to be noted. In my introductory remarks I stated that many factors must be considered in estimating the value of fistulization. This patient suffers from a nasal allergy. Every time there is an increase in the intensity of the allergy, there is a drop in the hearing acuity in both ears. If one studies Fig. 10, the audiometric recordings seem better, but as I remarked before, there is more to hearing than shown on the audiometric charts, or for that matter in the results of testing with tuning forks. In this case, with an open fenestration yielding a positive fistula reaction, hearing becomes worse during the attacks of allergy. Is this not further evidence that a permanently *open fenestrum* in the bony labyrinth capsule *is not the sole factor* in establishing and in maintaining better hearing acuity. I call your attention to the drop in the bone conduction in this case. I cannot yet explain this, since — as I will show further on — such finding is not incompatible with an excellent result for air conduction (Fig. 16).

I have other cases whose fenestrations still remain open, but which, after an incidental upper respiratory infection, have lost the newly gained hearing acuity acquired by the operation. Campbell makes a similar observation, but in some of my cases I find that the hearing has not regained its increase after the infection subsided, although the fistula has remained open. This, too, needs clarification.

In the case shown in Fig. 11, the hearing chart is that of a two-stage operation, based upon the combined technique of Sourdille and Lempert. The first stage consisted in the exenteration of the mastoid process, the removal of the bony external auditory canal wall, the freeing of the drum by removing the sulcus tympanicus, opening into the epitympanic cavity, and separating the articulation of the major ossicles, and removing the head of the malleus. The flap was then placed over the *unopened* semicircular canal, and the mastoid cavity was permitted to heal and epidermatize. The second stage consisted in elevating the thin epidermal layer over the mastoidal area, exposing the horizontal semicircular canal, fenestrating it, and then replacing the dermal flap. The shaded portion of the chart denotes the tests taken during the first stage. This shows a loss in hearing acuity for both the *operated* and the *opposite* ear. After the second stage was completed increased hearing in both ears for all tones is clearly evident. The operated ear is much better than the opposite ear. From all standpoints, this must be considered a successful outcome thus far.

In viewing Fig. 12, the great increase in the conversational range is striking. This 45 year old woman would hardly have been considered to hold out prospects for such a successful outcome. Her bone conduction, with masking, taken prior to operation for the second stage would certainly be a contraindication, if we are to accept the presently proposed guides.

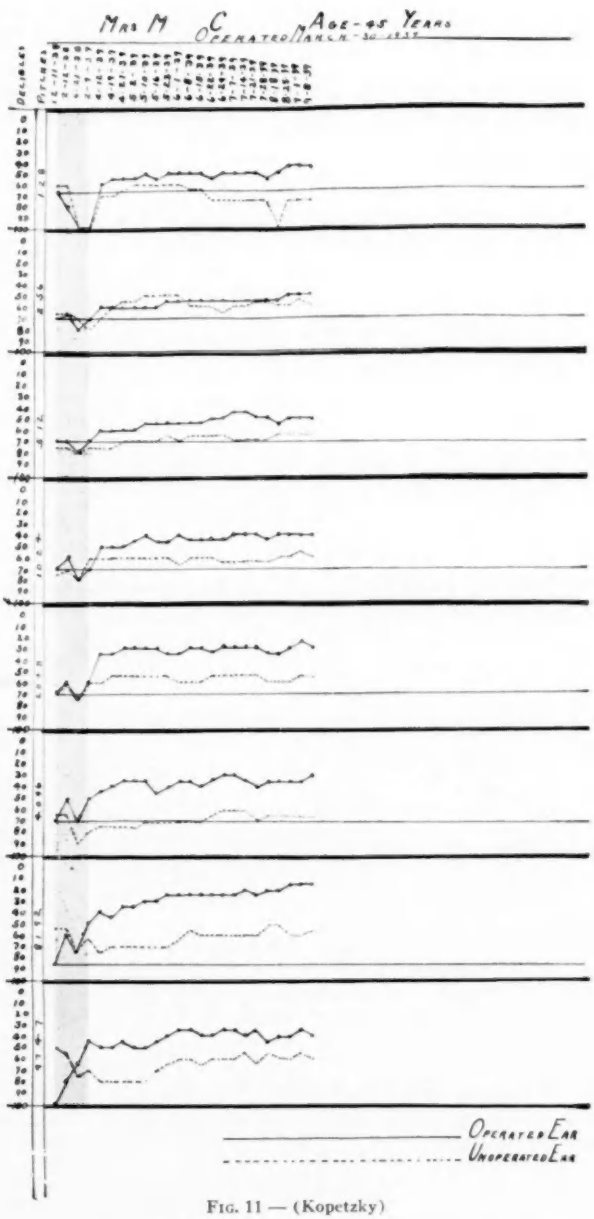
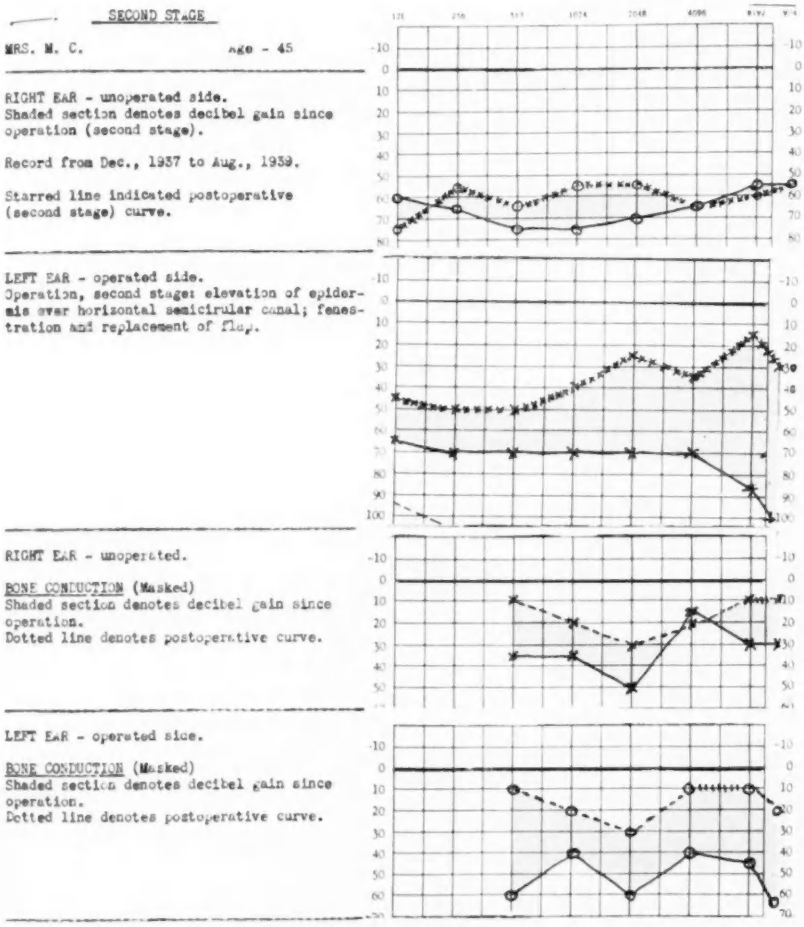


FIG. 11 — (Kopetzky)

NOTE. — The ear marked *unoperated* in the above chart, should be understood to mean, the ear which is not fenestrated. The opposite ear in this patient was operated upon, but did not have a fenestration done.


SECOND STAGE
MRS. M. C. Age - 45
RIGHT EAR - unoperated side.
Shaded section denotes decibel gain since operation (second stage).
Record from Dec., 1937 to Aug., 1939.
Starred line indicated postoperative (second stage) curve.
LEFT EAR - operated side.
Operation, second stage: elevation of epidermis over horizontal semicirular canal; fenestration and replacement of flap.
RIGHT EAR - unoperated.
BONE CONDUCTION (Masked)
Shaded section denotes decibel gain since operation.
Dotted line denotes postoperative curve.
LEFT EAR - operated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel gain since operation.
Dotted line denotes postoperative curve.

RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Dec., 1937	58%	56%
Aug., 1939	47%	31%

FIG. 12 — (Kopetzky)

But of greater moment is the fact that the improvement in her air conduction is *here paralleled by an improvement in the bone conduction (taken under conditions of masking)*, and the fistula remains open after a 7 months' period of observation. She not only hears well, but her awareness of her surroundings is better. We have no means of recording this. This case, as well as those shown in Figs. 3 and 4, and 5 and 6, prove conclusively that the permanence of the fistula is not achieved by the tissue which covers

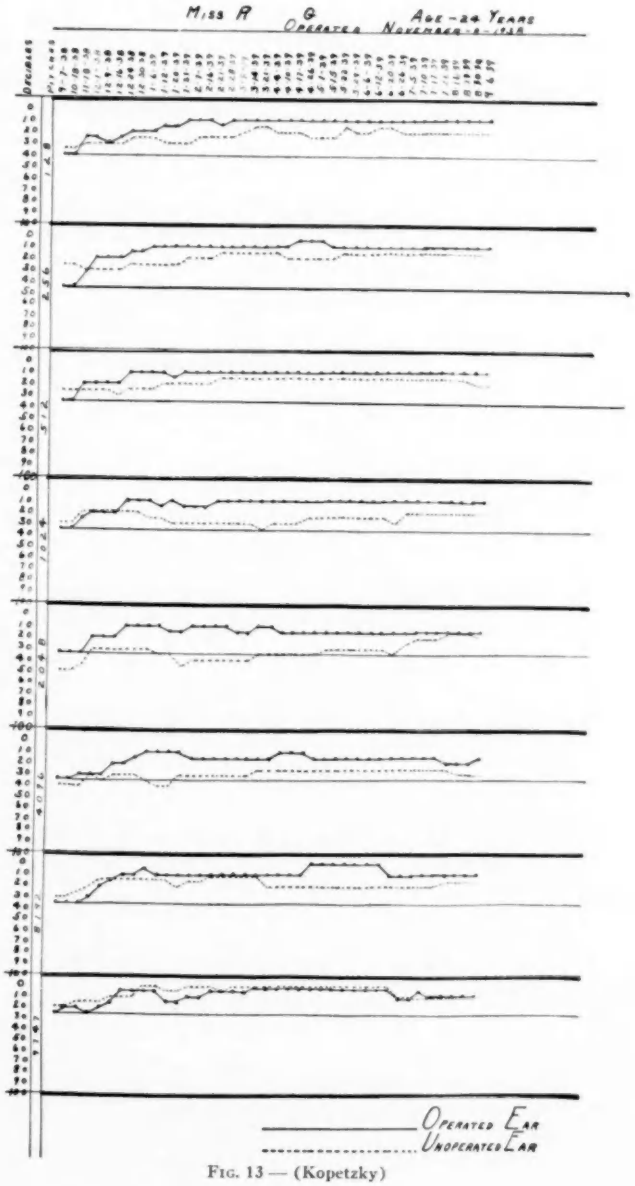
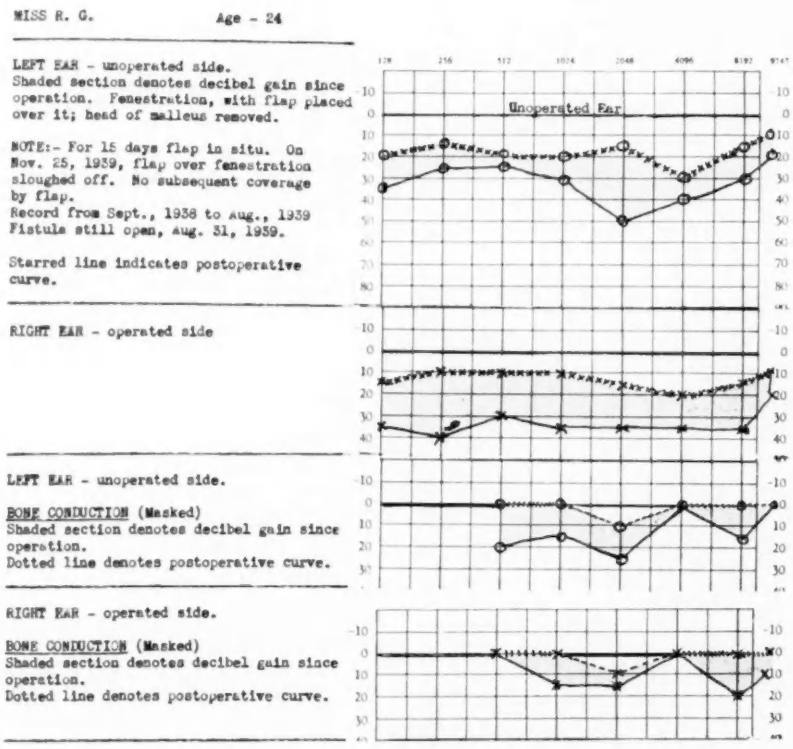


FIG. 13 — (Kopetzky)

it. What is necessary is that the tissue should eventually take on the characteristics of periosteum, and to hasten healing and secure a dry ear, be clothed by pavement epithelium.

Fig. 13 is the record of a one-stage operation, with resection of the head of the malleus, *maintaining the incus in situ*, and fenestration of the horizontal semicircular canal — using the posterior-superior portions of the membranous canal to cover the fenestrum. Note the gradual increase in


MISS R. G. Age - 24
LEFT EAR - unoperated side.
Shaded section denotes decibel gain since operation. Fenestration, with flap placed over it; head of malleus removed.
NOTE:- For 15 days flap in situ. On Nov. 25, 1939, flap over fenestration sloughed off. No subsequent coverage by flap.
Record from Sept., 1938 to Aug., 1939
Fistula still open, Aug. 31, 1939.
Starred line indicates postoperative curve.
Unoperated Ear
RIGHT EAR - operated side
LEFT EAR - unoperated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel gain since operation.
Dotted line denotes postoperative curve.
RIGHT EAR - operated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel gain since operation.
Dotted line denotes postoperative curve.

RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Sept., 1938	27%	28%
Aug., 1939	9%	15%

FIG. 14 — (Kopetzky)

hearing acuity for each tone. Note also the improvement in the unoperated ear for all tones. For the conversational range of 512, 1024 and 2048, the operated ear shows an impressive improvement; the opposite ear shows marked improvement only in the lower ranges, but little for the 1024 or the 2048 notes. (Fig. 13 follows.) There is improvement in the opposite

ear for the 4096 tone, and a marked improvement for the 8192 and 9747 tones.

A factor in this case deserves mention, namely — that the flap sloughed off after 15 days. Since that time there has *not been any covering* over the fistula except a blanket of granulations — which I am continually cauterizing with silver nitrate. The fistula has remained actively open for 11 months, and is still open. This case too, is still under observation and study. Her hearing in the operated ear is now within normal limits. The audiograms and the "breakdown" chart show this. Both ears are improved. For conversational tones (Fig. 14) the percentage loss is now only 9% in the operated ear, and 15% in the unoperated ear.

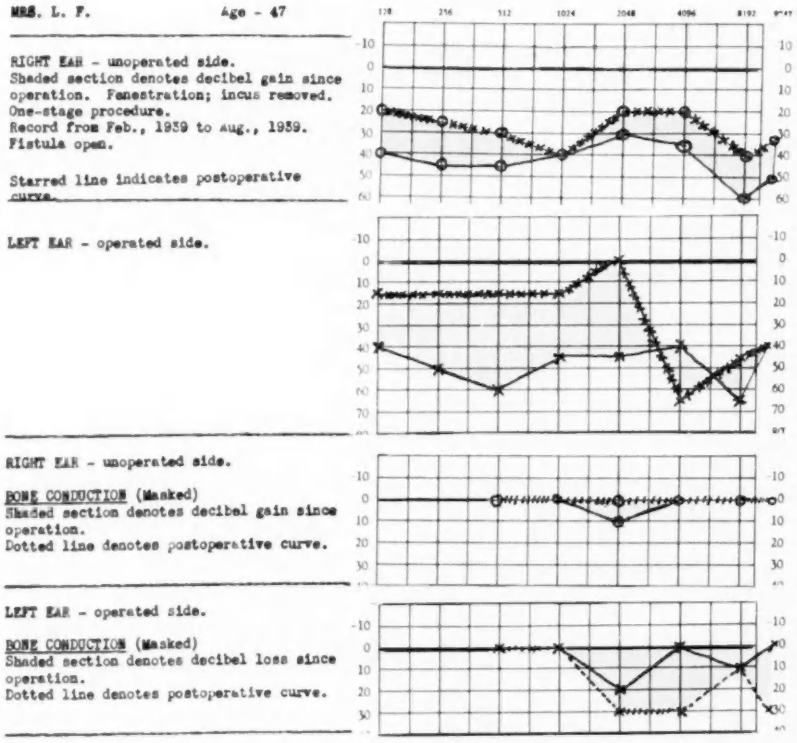
Where Campbell reports on the question of flaps, I ask — what about the necessity for a particular type of flap, about which there has been so much stress? This case is no accident. The permanent patency of the fistula here has shattered all my former beliefs concerning the importance of the covering membrane. There are definite factors in the *making* of the fistula which inhibit bone proliferation. When these are achieved a fistula will remain open, and hearing acuity will be increased in the given case — regardless of what covers the fistula.

Fig. 15 is a record of the hearing acuity of a case operated on in a one-stage operation, head of the malleus resected, the *incus removed*, and the posterior meatal membranous wall used to cover the fenestration. I call your attention to the striking increase in all tones up to and including 2048. I also call your attention to the improvement in the opposite ear.

There has been considerable discussion as to the need for preserving the incus, presuming it to articulate with an ankylosed stapes. Its utility as a factor in transmitting sound impulses, on the basis of any of the accepted theories by which we are supposed to hear, I seriously question. Certainly this case seems to indicate that its preservation is not a necessary factor to gain a great betterment in hearing acuity. That it may act, as Sourdille suggests, as a fulcrum over which sound impulses impinging upon the drum may be transmitted to the fistula along the tympano-meatal flap, I seriously doubt. The excellent results obtained in the case charted as Fig. 13 and 14, and in this case (Figs. 15 and 16) raise the question of the necessity for always retaining the incus, and for the absolute need of a flap for the achievement of hearing betterment by fistulization.

Note also in this case that in the highest tones (Fig. 16) no improvement has occurred, but nevertheless the improved hearing for the conversational range is so good that the percentage loss for these tones has improved from a 40% loss to one of only 9%. Nevertheless, as I have pointed out in Fig. 10, there has occurred a loss in the bone conduction (by masking) since operation. This too awaits explanation since this case is thus far a success from all standpoints. (Fig. 16 follows.)

In Fig. 17, which follows, I bring another point into the discussion. This is the hearing chart of a patient who for reasons having nothing whatsoever to do with the topic we are discussing, required an operation which embraced surgical destruction of the membranous horizontal semicircular


MRS. L. F. Age - 47
RIGHT EAR - unoperated side.
Shaded section denotes decibel gain since operation. Fenestration; incus removed.
One-stage procedure.
Record from Feb., 1939 to Aug., 1939.
Fistula open.
Starred line indicates postoperative curve.
LEFT EAR - operated side.
RIGHT EAR - unoperated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel gain since operation.
Dotted line denotes postoperative curve.
LEFT EAR - operated side.
BONE CONDUCTION (Masked)
Shaded section denotes decibel loss since operation.
Dotted line denotes postoperative curve.

RECORD OF CONVERSATIONAL LOSSES

Date	Right	Left
Feb., 1939	51%	40%
Aug., 1939	24%	6%

FIG. 15 — (Kopetzky)

canal. He made an uneventful recovery, did not develop any post-operative sepsis or labyrinthitis. His hearing acuity both before and after operation are here charted. Note that three months after the operation his hearing acuity in the operated ear was practically the same as it was before I destroyed the membranous canal. Can we not deduce from this and from other cases recorded in literature, that surgical trauma to the membranous labyrinth which is *not followed by infection,* need not always mean that hearing will be totally lost? Incidentally, from this finding we may also surmise that a prompt decapsulation of the membranous labyrinth, where an acute hydrops threatens the function of Corti's organ, may be indicated to avert the hearing loss threatened by the developing hydrops. On this point, too, further observations are necessary.

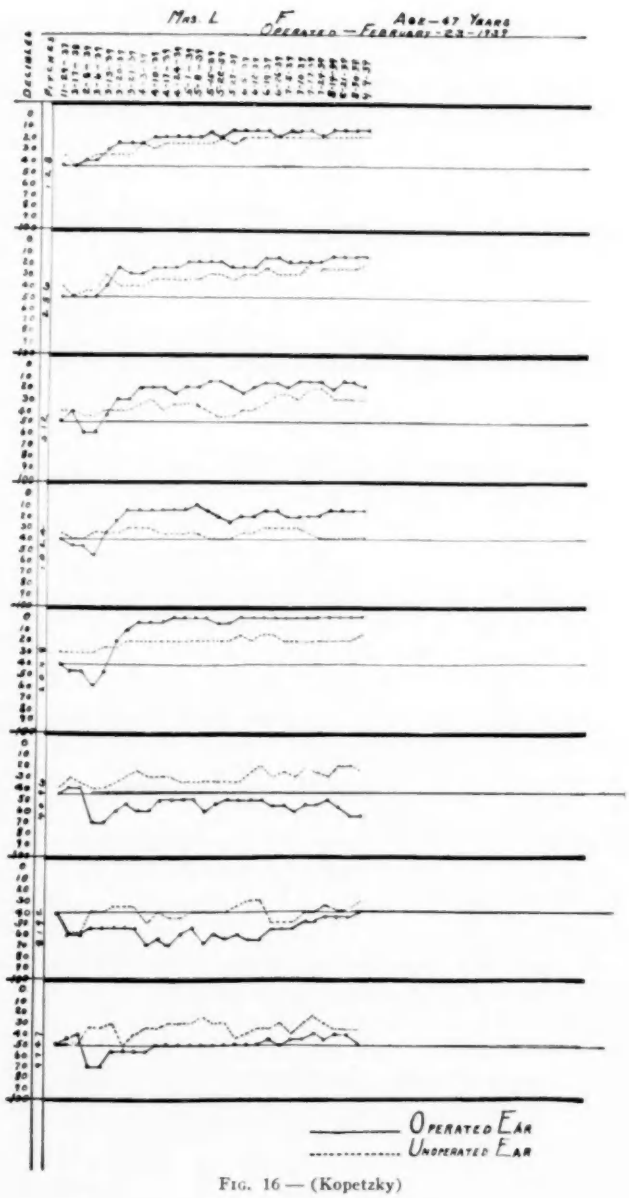


Fig. 16 — (Kopetzky)

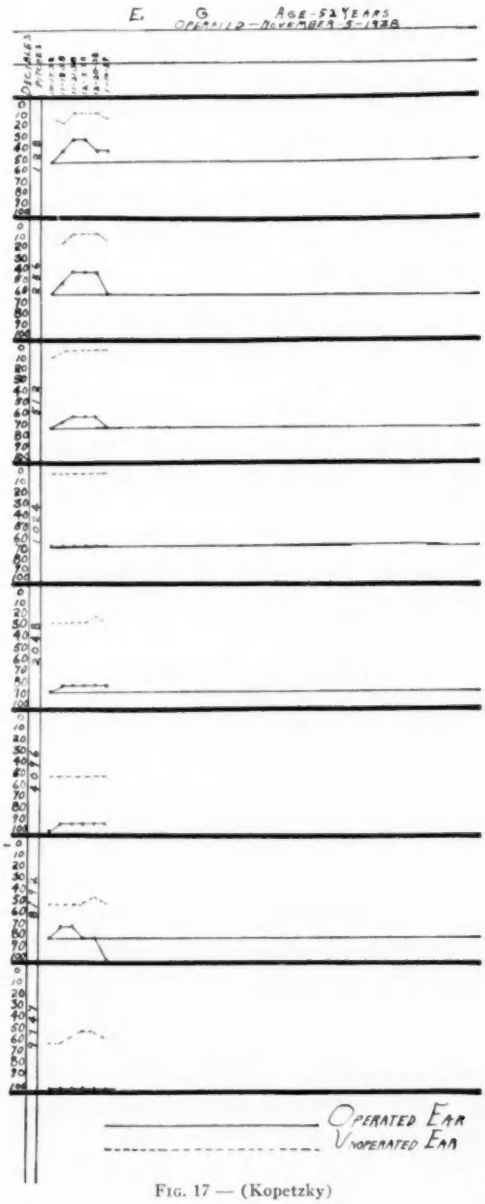


FIG. 17 — (Kopetzky)

COMMENT

From the material presented today by Campbell and myself, some tentative deductions may be drawn. I agree with Campbell and with Canfield, that the tissue destined to cover the fenestration is less important than the *manner of making the fistula.* Contrary to what has been said of the heat caused by the polishing bur, and the warning to avoid this heat, I believe that the heat which the bur produces is one of the most important elements in inhibiting bony closure of the fistula. In some of my best results, as far as maintaining a patent fistula is concerned, I so worked the bur that sufficient heat was created to produce a thermal labyrinthine reaction evidenced by induced nystagmus, long before the tell-tale dark line had brought the endosteal layer of the canal into view. Where, in the earlier cases, I carefully tried not to produce heat, my results were not as good as when I disregarded this warning and actually strove to produce a degree of local heat with the bur.

The size of the fenestration plays a part too, in the end-results. In a few cases, I made a long exposure of the membranous labyrinth. My results here were not good. My best results have been obtained with a fenestration of from one to three millimeters in length. Definitely, further study is required to determine the extent of the fistula needed in relation to th size of the round window, and to the degree of stapes fixation which is present in a given case. Furthermore, in some instances, even with a persistently open fistula, the hearing gradually drops, and the initial gain is lost. Here I have found that a systemic infection of one or another sort, was the inciting cause. Is there not some suggestion here that the causal incident which precipitated the deafness will continue and remain the factor responsible for the defeat of a successful fenestration?

I have presented Fig. 17 to demonstrate that deliberate destruction of the membranous canal need not of itself destroy or reduce hearing. As a matter of fact, in such an event a primary increase in hearing acuity often occurs — only to drop to its previous level. Finally — the data accumulated in my experience tends to bring into question all our accepted theories of the physiology of hearing. The collection of sound wave impulses by the drum, their transfer through an ossicular chain, their impact upon the labyrinthine fluids, and then a selective action by a given portion of the organ of Corti — all of this must be reviewed. Neither does the "telephone" theory of hearing supply the answer. The question of an electrical potential in the cochlea which initiates an action-current through the cochlear branch of the eighth nerve also falls short of entirely fitting the circumstances presented in these improved cases. Yet I feel that this theory, now under more intensive scrutiny, may hold the answer to our problem.

Diagnostic criteria in habitual use do not necessarily make for a positive diagnosis. In several instances, I have uncovered totally unsuspected pathology. When such is found it must be attacked first. The further results from fistulization then depend upon the resourcefulness and ingenuity of the surgeon.

I have called attention to the behavior of the unoperated ear in these cases. It deserves study. In my cases wherein a cholesteatome impeded hearing, the unaffected ear was normal. Where, however, a chronic progressive

deafness was present, both ears gradually lost hearing acuity. When, in such cases, a successful fistulization had been accomplished, not only did the operated ear show improvement, but the unoperated ear also. Is it not therefore important to revaluate the bilateral innervation of the chochlea, and its central distribution? Can we not conceive that the awakening of the cortical hearing centers, stimulated by the re-activation of the one ear, through afferent nerve impulses, will increase sensory perception by the unoperated ear? This finding, to my mind, opens up the entire question of the role which the brain as a whole, as well as designated parts of it, plays in the function of hearing.

I feel that before operating on these patients, great care should be exercised to learn which ear was first involved in the deafness. That probably is the ear at fault, and I am strongly impressed that the second ear has lost function largely because there has been a lack of stimulation to the association centers over a long period of time, from the initially involved side. I have records of very early cases, where only one ear showed the classical symptoms of otosclerosis, and only after an interval of some years did the second ear follow suit. This I have observed particularly in some very young patients.

In closing these brief hints *on the problems involved,* suffice it to say that the problem of affording relief surgically to the deafened is still far from solution. I strenuously urge that the surgical work be continued. We shall learn much from careful observations made by competent surgeons. Above all, I trust that physiologists will re-examine, in the light of the data obtained by this surgery, the fundamentals of hearing.

On the question of what permanent value this surgery holds out for the deafened, no final conclusion can be made as yet. More time must elapse. At present, there seems to be reason to hold to the view that something is developing which will be of value in the therapy for deafness.

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DISCUSSION

JAMES A. BABBITT, M.D., Philadelphia: In 1871, Kessel of Jena, who had been studying the problem of the relative necessity of the tympanic membrane and ossicles in hearing, began operating in cases of deafness and subjective noises removing adhesions and even the stapes, and contributed valuable papers in 1875 and later reported 16 successful operations. Schwarze, influenced by the fact that the malleus and incus were frequently discharged during chronic suppuration, without serious loss in hearing and supported by Kessel's work began such surgical attack.

Development in method occurred during years following, associated with the work of such well known men as Hartmann, Neuman, Pollitzer, Heath and Kashiwabara. Fritz Thies, Senior, performed the first radical operation through the external canal in 1907 and followed this method in 1500 cases without casualty. You are doubtless acquainted with the work of our own George Tobey, who published an account of his own Endaural approach in 1930. The same year Komuro reported on a modified attic drainage, later developed in this country.

Without further chronologic detail, the Endaural approach in acute and chronic suppuration has been developed by Lempert, cited by the speaker in this paper. He has reported some 1800 operations on the mastoid and middle ear through a triangular incision which makes an extra cartilaginous movable window. His success has influenced many toward the Endaural route in both acute and chronic infections. From this work developed fistulization in this country for otosclerotic and other types of progressive deafness.

The work of Holmgren in Sweden and Sourdille in France has already progressed far toward a successful surgical status in deafness, but by a post auricular and usually a two-stage operation.

It has seemed best to review this background to correlate the present advance reported in this paper. The choice of route, Endaural or Post Auricular, is not of course of such vital interest as the success in maintaining the labyrinthine fistula.

It is gratifying to have such an important otologic topic presented with such frankness and meticulous care. In addition to his frank report, Doctor Campbell has presented a broad point of view in respect to this important field of advance.

Perhaps the work of Doctors Duel and Ballance has accomplished much in stimulating the present day interest toward ferreting out the niceties of this rather drab type of ear research. For the past two years your Chairman and others have sponsored and endeavored to stimulate younger operators of ability in developing the technique of this procedure and entering into the fascinating phases of its research. One brilliant operator in this group has just presented to you his preliminary results and while they represent but four successful out of the first eight cases, the reasons for failure have been honestly presented and any of you who have watched one of these operations during its 3 or 4 hour period, will realize how tedious and delicate it is.

As suggested by Doctor Campbell, the actual obtaining of immediate hearing from fistula in any part of the labyrinth has been long known but the maintenance of this fistula opening for any appreciable length of time is only in the initial stage.

Much research remains to be done. The physiologic reason for this quick response of hearing is still problematic and a matter of conjecture. Its relation to tinnitus is even more difficult, and this may shake the long standing assumption of the relation of certain types of tinnitus to cochlear degeneration. Perhaps the whole interpretation of otosclerosis as a familial entity has come to the bar of judgment.

If the findings of Doctor Crowe and his associates in Baltimore are substantiated, the Gray theory of familial descent may be altered and the treatment and surgery for this condition be increasingly relegated to the middle ear. The whole problem of relief for deafness may demand a realignment in the correlation of values of biochemistry, naso-pharyngeal preventive measures, hearing aids of different types, blocking of the round window and surgical fistulization.

Many things enter the scale of readjustment under the fistulization procedure, such as the percentage of bone conduction justify operation, the relative decibel value of the hearing aid and its possible contribution to future increase in deafness, and the guarantee of vestibular integrity. As suggested above, the question of post-auricular or endaural approach is not so important as the skillful technique upon reaching the labyrinth, and the management of granulations and sepsis in the post-operative field.

After all, in the final analysis perhaps something else must be considered, and that is the final reaction of the patient from operative shock and his acceptance of physiological and psychologic conditions.

This has been a fine paper and the more frequently such efforts are carefully reported, the sooner we shall reach a justification for fistulization surgery.

JULIUS LEMPERT, M.D., New York: I have performed the fenestration technique on 115 patients. They may be divided into two classes: Suitable cases with good bone conduction and conversational frequencies of 512, 1024 and 2048, and non-suitable cases with poor conduction in the same frequencies. Seventy-six suitable cases were operated upon. Of these, 65 resulted in a permanent restoration of practical hearing. These 65 patients are now socially and economically rehabilitated. Thirty-nine non-suitable cases were operated upon. Of these 10 resulted in good hearing improvement but not practical hearing.

In every case where a permanent restoration of practical hearing was obtained following fenestration, tinnitus completely disappeared on the operative side. In those cases where hearing improvement did not result from fenestration, tinnitus remained, and in those cases where hearing which had improved began to recede for various causes, tinnitus gradually began to come back and reached its height again when complete closure took place.

Although permanent restoration of practical hearing was obtained in only 75 cases, the newly-created fenestra remained permanently open in 96 of the 115 cases operated upon; thus in 21 cases hearing improvement was not obtained in spite of the fenestra remaining open.

I have inspected and revised a newly-created fenestra of the external semicircular canal in 28 of the 115 post-operative cases. In 4 of these cases the fenestra was revised twice and in another case three times; thus, 34 revisions were performed on the 28 cases. Of the 28 cases revised new bone formation was observed in only 19 cases.

I want to congratulate Doctor Campbell on his observations and his good results.

I should like to state that Doctor Campbell in his indications stated that previous middle ear infection or a perforated drum is not a contraindication. In my experience I have found it to be a distinct contraindication. I do not think it is advisable to operate any case where there is even a history of previous middle ear infection, for two reasons. First, that the operation, if intratympanic surgery is involved, may reawaken a dormant pathogenesis which may be existing, or even if there is no such thing existing, the chances for success are not always good, and at least if not good, they are absolutely unknown because we do not know the status of round windows in cases where there was a previous infection. There may be an existing fibrosis of round windows, and in order to obtain a permanent result in hearing, or restoration of practical hearing, it is essential to have two windows, both normally functioning, in order to be able to reestablish a free mobility of the perilymph and the endolymph by air-borne sound.

An improvement in hearing in these cases, unless it is of practical value to the patient, is not worth the effort of operation. The patient is not interested in whether his audiometric reading is 70 decibel loss by air or 50 or 40 decibel loss. The patient is interested in whether we have brought his hearing back to the practical level, whether that be 20 or 30 decibels or whatever it requires. That is the interest of the patient, and in order to obtain a practical hearing improvement, it is most important not only to perform the gross steps in the technique, but to pay the strictest attention to the technical minutia which cause defeat in the most perfectly performed operation if one is not careful in attending to the least detail in this operation.

As to the time of the complete closure of the fistula, whereas a fistula may not close completely within two and whereas it may take four months for complete closure, evidence of closure is inevitably observed by the diminution in the vestibular response to the fistula test as early as the third or fourth week postoperatively. There is a diminished response. The minute this diminished response is observed, this is a sign that new bone formation or fibrosis is taking place and that is a sign that the fistula is beginning to close. Whether it takes three months for complete closure is immaterial, but if the fistula did not show signs of beginning closure within two months postoperatively, it did not close in any of my cases.

Doctor Kopetzky has stated his experience with these cases, and he stated that in several cases he left the fistula completely open without covering it with a membrane, because the membrane sloughed or for some other

reason and in spite of that the fistula remained open and hearing was maintained. That has not been my experience. I have deliberately left fistulas open and have not covered them with membrane in cases where there was no danger to the patient's hearing, because the patient from the start was a contraindicated case, and had poor bone conduction. In every one of these cases fibrosis closed the window, if not bone formation. It does not matter whether it is new bone formation or fibrous tissue which affects the closure of the window. The fact remains that the window is closed and there is an impedence created to the transmission of air-borne sound and to the mobilization of the perilymph and the endolymph; whether it is through fibrosis extended from the mastoid wound into the fistula or whether it is due to closure by bone does not matter. That has been my experience and it has been the experience of men like Doctors Sourdille and Holmgren, who have been doing this work for twenty-five years, that a fistula left open closes no matter what else happens.

EDWARD H. CAMPBELL, M.D., in closing: I have just two or three words to say.

(Slide) Doctor Lempert spoke about the contraindication. He expressed the thought that an ear that has previously been the subject of otitis media or inflammatory changes is not a good case for operation. As a rule that had been my impression, too, until I operated on this patient (it was my sixth case) and the patient had had numerous middle ear abscesses and the drum was very much thickened, retracted, scarred, and showed a healed perforation.

Here is her audiometer reading before operation. The operation was done just a year ago. This audiogram shortly before the operation showed 61.6 per cent loss of hearing. This is something that I have observed in all the cases I have done. The improvement reaches its greatest point about a month following operation. About a month after operation the hearing was improved 30 per cent. You see the differences in the decibel readings at these different frequencies. In spite of the history of previous middle ear troubles, this shows a very marked immediate improvement. Another characteristic feature is that the greatest amount of improvement, usually about a month or six weeks after operation, is not maintained, even though the fistula does not close by bony regeneration. In another two or three weeks, as this slide shows, there had been some loss; in another month about the same loss, and slightly more one year after operation, which I assume will be the final result. I have tested her since then and there has been no further change. This leaves a 20 per cent improvement in hearing, which has been maintained. The fistula remains active and the improvement has been maintained in spite of previous history of middle ear abscess.

The other ear had 49 per cent loss before the operation on this ear and the hearing loss in it has been practically the same throughout.

I wish to thank Doctors Babbit, Kopetzky, and Lempert for their kind discussion.

SINUSITIS FROM THE VIEWPOINT OF THE GENERAL PATHOLOGIST

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BY INVITATION
WASHINGTON, D. C.

THIS STUDY was made on the material from 150 cases in the Academy's Otolaryngic Registry at the Army Medical Museum. It must necessarily be a morphologic one because first I am not a clinician and secondly we have little or no clinical data on many of the cases. I am afraid, therefore, that I cannot bring a great deal that would be of practical value to you but there are several features in the material that appeal to me as a pathologist and which have not been particularly emphasized in previous discussions of the pathology of sinusitis. These I propose to speak of particularly rather than re-hash the general details that have been covered many times before in the literature. I have excluded from the study the specific granulomatous infections and neoplasms.

There are only about a dozen elements involved in the reaction of tissues to injury, be they the sinuses, the appendix, eye or whatnot, but there are so many factors that modify the combination of these elements that the ultimate pictures may be quite different and complicated. It is the pathologists' business to unscramble these pictures and interpret them in terms of etiology, prognosis, etc. In the case of the sinuses there are indeed many factors. We have the general ones, the patient's occupation, age, the environment in which he lives, particularly climatic conditions, the general personal factors such as habitus, which I consider of great importance, the degree of resistance to infections, the existence of allergic states, his diet, the sensitivity of his vasomotor mechanism, and finally the local conditions in the sinuses themselves. If the sinuses *are* vestigial they share with such structures the lower vitality, resistance and regenerative powers, although we do know that the sinus mucosa will regenerate satisfactorily if conditions

* Curator, Army Medical Museum, Washington, D. C.

are appropriate. The mucosa of the sinuses differs, morphologically, very little from the Schneiderian membrane of the nose but it is certainly much less active physiologically. There is, of course, the difference in vascularity and gland content and these have their influence on the results of injury. The mere fact that the sinuses are cavities with such small drainage facilities that are so readily occluded is a very important factor, as are also the anatomical variations incident to age, and the individual. With these you are more familiar than I.

Classification, of course, is the ultimate aim of all scientific endeavor and I suppose that eventually some satisfactory classification of sinusitis will be evolved but it certainly has not been done to date. It is confusing and somewhat amusing to go through the literature and see the efforts that have been made to devise a classification that would be scientific and practicable. Much of the writing has been done by clinicians with not too much background in pathology who have not always stopped to define their terms so that there is a mixture in terminology that adds to the confusion. The modern tendency in pathology is to classify on the etiologic basis so far as possible. The modern pathologist does not speak now of Laënnec's or Hanot's cirrhosis of the liver but of biliary cirrhosis or alcoholic cirrhosis. One of the things that occurred to me as I went through this group of cases was that a classification on the basis of etiology might be possible from the histologic picture. I have divided the cases tentatively into three groups:

INFECTIOUS, which would include those incident to trauma;
ALLERGIC, which would include all cases incident to vasomotor phenomena;
MIXED, which usually means that they were primarily allergic and subsequently complicated by infection.

TABLE I

Distribution of 150 Cases of Sinusitis on an Etiologic Basis

Infectious	75
Allergic	28
Mixed	47

The case is considered allergic even in the absence of clinical history by the high percentage of eosinophiles, polypoid edema, and thickened basement membrane. It is not meant, of course, that

the presence of one or two eosinophiles means allergy and the picture varies with the stage of reaction as has been so well shown by Steinberg.[1] We have the two types, therefore, the pure in which the history and picture are decisive, and the presumptive in which there may be history of hay fever or asthma and histologically, arbitrarily say 30 per cent or more eosinophiles.

There is, however, a certain amount of practicability in a straight morphologic classification. The clinical course and treatment of a suppurative sinusitis would certainly differ from that of a polypoid and the two would not be comparable to the low grade chronic indurative type of reaction. I have made, therefore, a classification on this basis. Table II shows the distribution of these forms in the three etiologic groups.

TABLE II

Distribution of Pathologic Types

	Infectious	Allergic	Mixed
Hyperplastic	36	1	7
Polypoid	14	20	24
Fibrous	20	9	15
Ulcerative	6	1	1
Hemorrhagic	8	..	3
Follicular Nodules	16	8	13

HYPERPLASTIC, with abundant mononuclear exudate, chiefly plasma cells, fibroblastic and reticulum increase and increase in young blood vessels;

FIBROTIC, very little exudate, much fibrosis of stroma—scarring, decrease in vascular elements and possibly also in number of glands;

PAPILLARY, usually seen in the hyperplastic in which superficial edema and exudate form gross or microscopic papillary thickenings in the mucosa;

POLYPOID, *Hemorrhagic, Ulcerative,* which are self descriptive;

CYSTIC, Glandular — retention cysts of gland elements:

a. Mucous and serous.

b. Pseudoacini.

Interstitial — lakes of edema in the stroma, possibly also lymphangiectasis;

FOLLICULAR. In this I am attempting to introduce a new term comparable to the follicular cystitis of the urologists. It is based on the presence in the mucosa of lymphatic nodules and will be discussed later.

One of the striking things about the picture is the rarity of a real suppurative type of exudate in the sinus stroma even in those cases which clinically are purulent. Collections of polymorphs may be found in dilated glands and occasionally an interstitial abscess is encountered but the predominant cell in the infectious group is the mononuclear, particularly the reticulum and plasma cell. It makes one wonder if bacteria play such an important role after all and if, as Fenton[2] has written, the less tangible factors as environment, vasomotor sensitiveness, diet, etc., may not be of greater importance in the non-purulent cases.

From the distribution of these types in the three etiologic groups it is evident that the hyperplastic reaction is more common in the infectious group and the polypoid in the allergic. That is about as far as it goes and it would seem to be more practicable at least from the pathologist's standpoint for the report on the specimen to include the pathologist's interpretation of the etiology and to modify this with the type of reaction because of possible aid in determining treatment. Even at that, the experienced clinician can size up his case and determine the course of treatment with greater surety than can the pathologist, after eliminating neoplasm.

I wish now to emphasize particularly the follicular form. In thirty-seven, or 25%, of the 150 cases reviewed lymphatic nodules were found in various stages of development. There is nothing new, of course, in the recognition of these follicles. They were specifically mentioned almost 50 years ago by Zuckerkandl,[3] and they have been mentioned by a number of writers since, especially by Semenov[4] in his comprehensive writings of the past few years, but no one has attempted to determine their significance nor to recognize them as a specific entity as has been done with almost every other element in the pathologic picture. This is my only claim to originality, that is, in adding follicular sinusitis to the already overburdened list. I don't know that I am contributing much in doing this because aside from the fact that they are a feature of the chronic cases and they occur less commonly in the pure allergic I do not have sufficient clinical data to determine, for example, what significance their presence is in terms of prognosis or treatment. We see them in chronic inflammations involving other parts of the body. Your ophthalmologic colleagues are familiar with them in the conjunctiva in vernal catarrh and trachoma.

TABLE III

Incidence of Lymphatic Nodules	
Infectious	16
Allergic	8
Mixed	13

Table III shows the dominating influence of infection in their production. I would be interested to know, for example, whether their presence would presage a favorable response from radiation therapy or if malignancies occurred more commonly in the mucosae showing them than otherwise. I have in mind the comparable condition in the bladder. In reviewing a thousand or so bladder tumors recently I was impressed by the frequency of the follicular cystitis as a concomitant. It is hardly possible to say, however, whether the lymphatic nodules were primary or developed as a result of the tumor.

TABLE IV

Anatomic Distribution of 111 Lymphatic Nodules Observed in 37 Cases	
Subepithelial	21
Stroma	68
Subglandular	13
Periosteum	9

Some of those who have written about the nodules stated that they were predominantly in the deeper layers of the mucosa in or near the periosteum. Table IV shows the distribution of the 111 nodules observed in the 37 cases. They are predominantly in the upper layers, many of them in direct contact with the surface or gland epithelium. Some have raised the question as to whether these structures are really lymphatic nodules and whether these central clear areas are really germinal centers such as those seen in orthodox lymphatic tissue. I am convinced that they are.

Fig. 1* (Neg. No. 68878 — Acc. No. 45790) — Male, age 46 years. Frequent head colds; nasal obstruction and discharge; headaches for two years; bilateral radical antra show pus and thickened membrane. This is a section of mucosa showing the 3 stages in the development of the nodule. There is first a diffuse

* All illustrations are from the Army Medical Museum files.

FIG. 1 — (Ash)

FIG. 2 — (Ash)

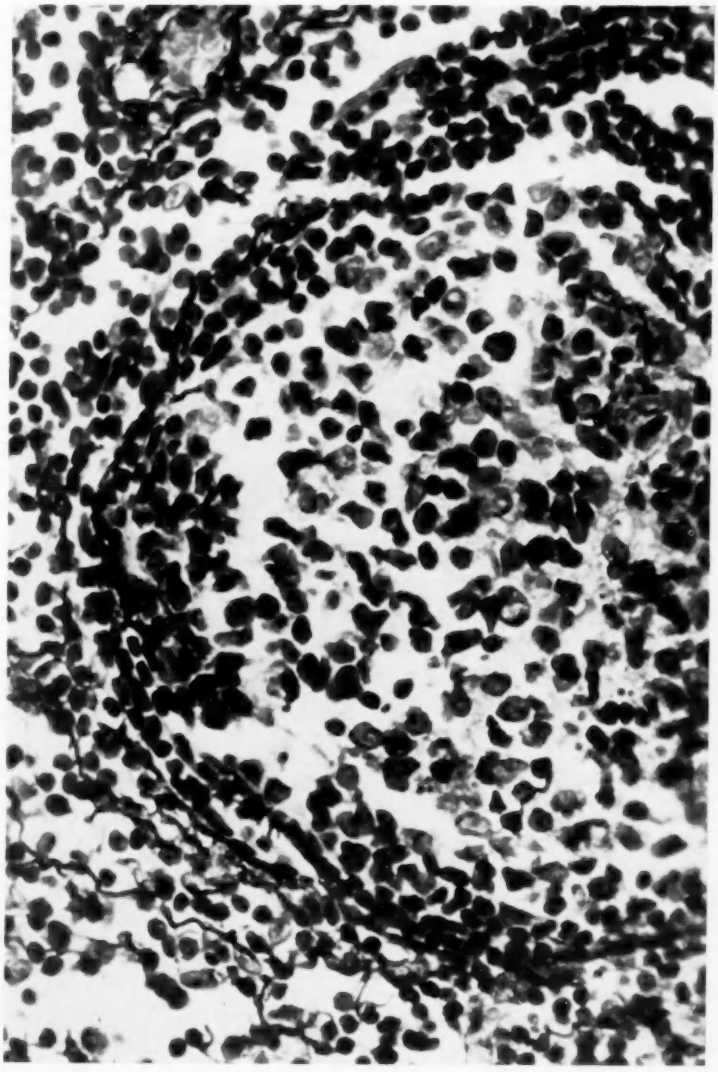

FIG. 3 — (Ash)

concentration of lymphocytes (a) about an artery that excludes practically all the local elements. Later the group becomes compact (b), more sharply circumscribed, a stage resembling the solid secondary nodule of normal lymphatic tissue and the blood vessel about which the nodule formed seems to be pushed to the periphery. Still later we have the appearance of the germinal center (c) and the nodule has all the characteristics now of the secondary Flemming nodule of normal lymphatic tissue. It has been claimed by some that these are really not germinal centers but collections of reticulo-endothelial cells and that the nodule should therefore not be called lymphatic but lymphoid. The reticulum stains, however, convince me that morphologically, at least, these are lymphatic rather than lymphoid.

Fig. 2 (Neg. No. 68877 — Acc. No. 64542) — Male, age 44 years. Chemical worker for 10 years; purulent nasal discharge, headache, blocked nose for three years. Pan-sinusitis demonstrated by X-ray. Bilateral Lynch; pus and polypoid tissue in frontals. A nodule in the compact stage shows a few thick fragments of the original reticulum of the neighborhood and a beginning reticulum meshwork about the central area.

Fig. 3 — Same case as Fig. 1. The mature nodule in which there is a total absence of reticulum in the germinal center and a well defined demarcation of the lymphocytic portion with its reticular meshwork, a characteristic of normal lymphatic nodules.

I am not going to open the discussion of the physiology of the so-called "germinal centers." The modern tendency is to get away from Flemming's original notion that the lymphocytes were actually formed by these large central cells, and to consider the centers as a part of the defense mechanism. If so, there would seem to be some logical reason for their presence in chronic inflammations. When once formed they seem to be an independent unit in the picture, however, for they will persist after the subsidence of the inflammatory phenomena.

Fig. 4 (Neg. No. 68889 — Acc. No. 46221) — Female, age 21 years. Frequent head colds; left nasal block; headache 6 to 7 months; left antrum lining removed. A nodule, rather concentrated, developing along an arteriole.

Another feature that impressed me in this material was the enormous amount of reticulum in the stroma of these chronically inflamed membranes.

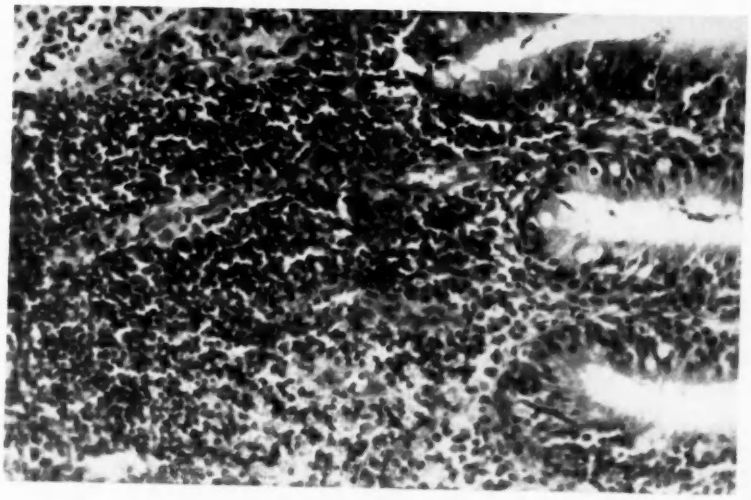

FIG. 4 — (Ash)

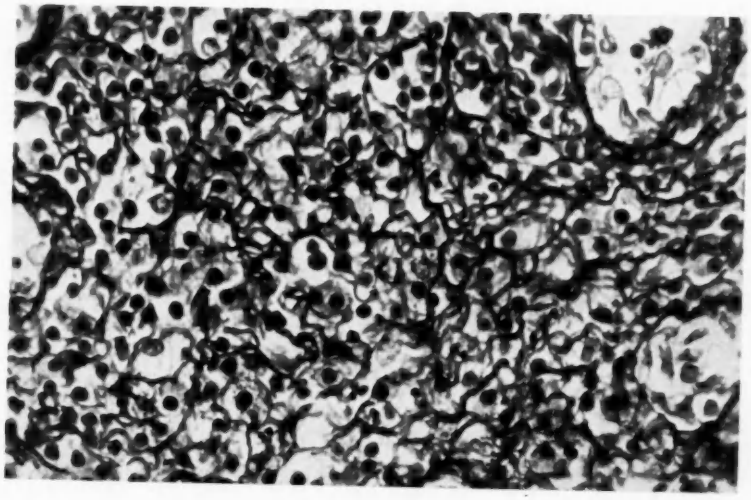

FIG. 5 — (Ash)

Fig. 5 — Same case as Fig. 2. Such a one stained by the Wilder silver method to demonstrate the fibrils. It will be noted that practically every cell is surrounded by a fibril. This is not an isolated field but the general picture through the stroma in those cases in which edema is not too prominent a feature. This indicates the prominent part that the reticulo-endothelial cell plays in the picture, particularly in the hyperplastic cases.

The next point that I wish to make is the histogenesis of the thick basement membrane.

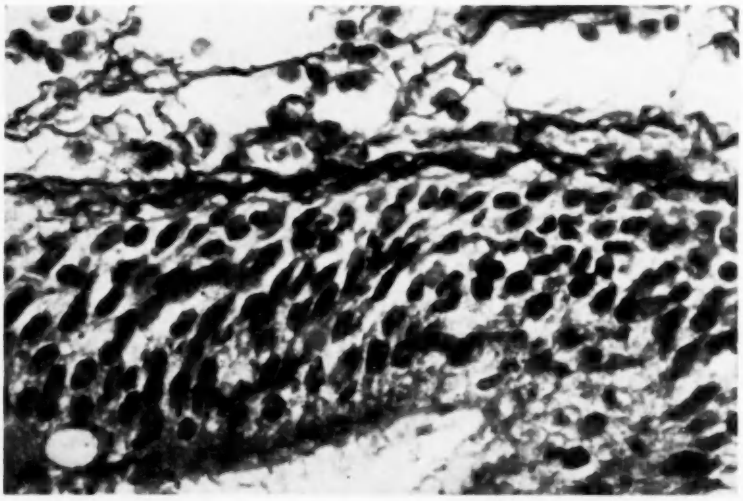

FIG. 6 — (Ash)

Fig. 6 (Neg. No. 68882 — Acc. No. 53119) — Male, age 40 years. Had tooth extracted 2 years ago when floor of antrum was penetrated; one year later developed nasal block on left side; maxillary sinusitis of long standing; left radical antrum; debridement on mucous membrane; antrum filled with polypoid and hyperplastic tissue and small encapsulated abscesses; no allergic history. Shows an early stage in which the thickening is barely recognizable in the hematoxylin and eosin stain. This is another silver stain to demonstrate the reticulum fibrils and it will be noted how they are concentrated just beneath the epithelium and how beneath this concentration there are large reticulo-endothelial cells surrounded by fibrils.

Fig. 7 — A thicker portion of the same membrane showing greater concentration of the fibrils. Some of the cells are being

compressed by the surrounding fibrils. Note also the relationship of these cells and fibrils to the blood vessels which are so near the surface.

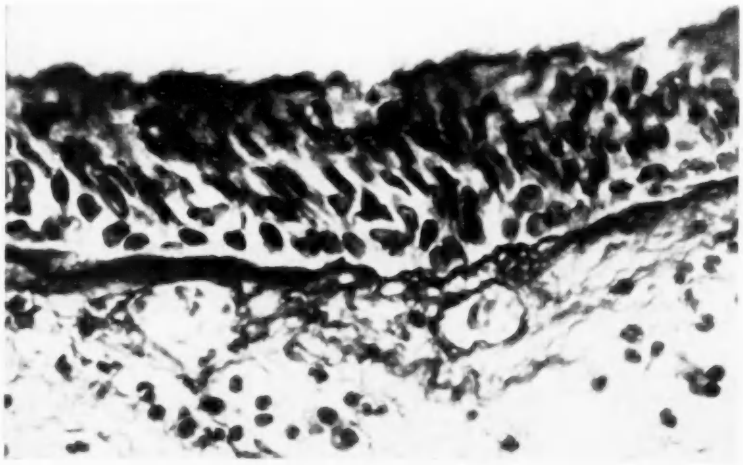

FIG. 7 — (Ash)

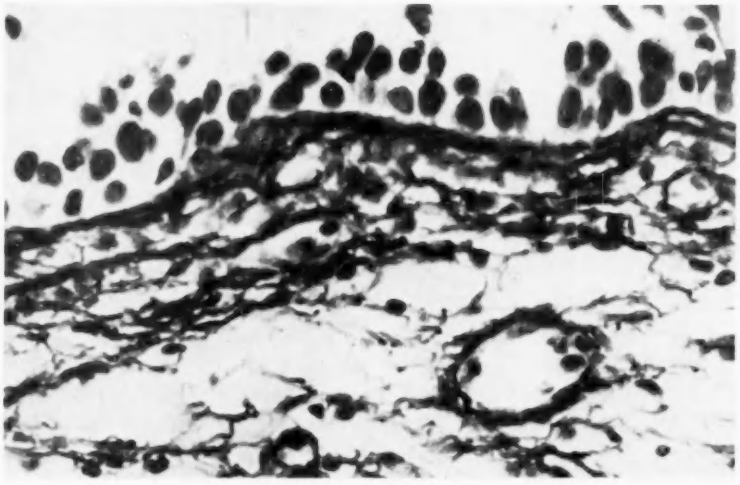

FIG. 8 — (Ash)

Fig. 8 (Neg. No. 68886 — Acc. No. 38173) — We have no clinical history. A still thicker membrane in which the fibrils are beginning to lose their identity and become fused into hyaline strands, the enmeshed cells disappearing in the superficial layers. Much of the surface epithelium is abraded.

Fig. 9 (Neg. No. 68921 — Acc. No. 52157) — Male, age 25 years. Swelling in the region of left eye for 2 years, not tender until 5 days before operation; occasional post-nasal discharge, hemorrhagic and mucoid in character; no allergic history; X-ray shows some density in both antra; radical frontal sinus; mucosa markedly turgescent; hyperplasia occluding naso-frontal duct

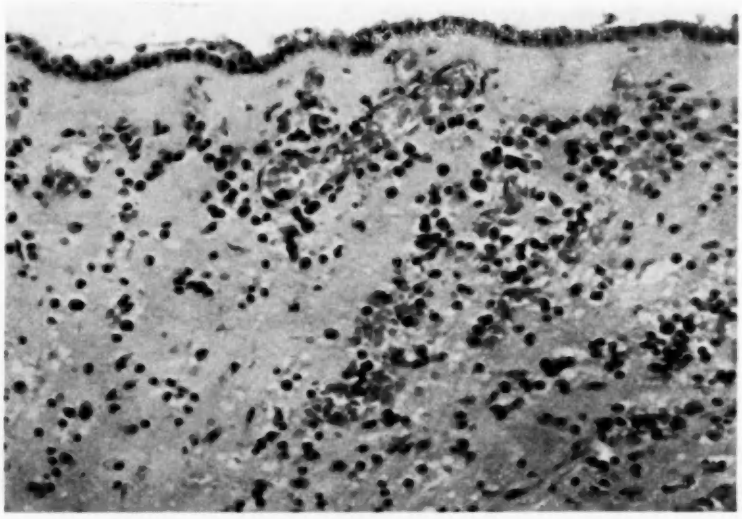

FIG. 9 — (Ash)

causing expansion of frontal wall. Malignancy had been suspected clinically. Hematoxylin and eosin stain of a mucosa in which basement membrane is enormously thickened and hyalinized and merges imperceptibly with the underlying fibrosed stroma.

Fig. 10 — A reticulum stain on such a field shows the preponderance of the hyalinized material over the fibrillar and the complete destruction of the reticulum cells. The surface epithelium is gone except for an occasional hardy leaf still clinging to the bough.

Some have interpreted this great thickening in the basement membrane as a defense mechanism. I do not believe that such inert material as this is much of a substitute for healthy epithelium whose major function is protection. This tissue has the fraility of any other scar tissue.

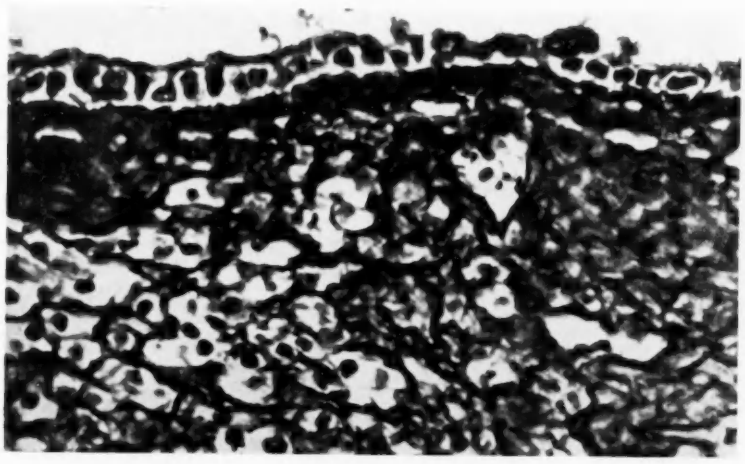

FIG. 10 — (Ash)

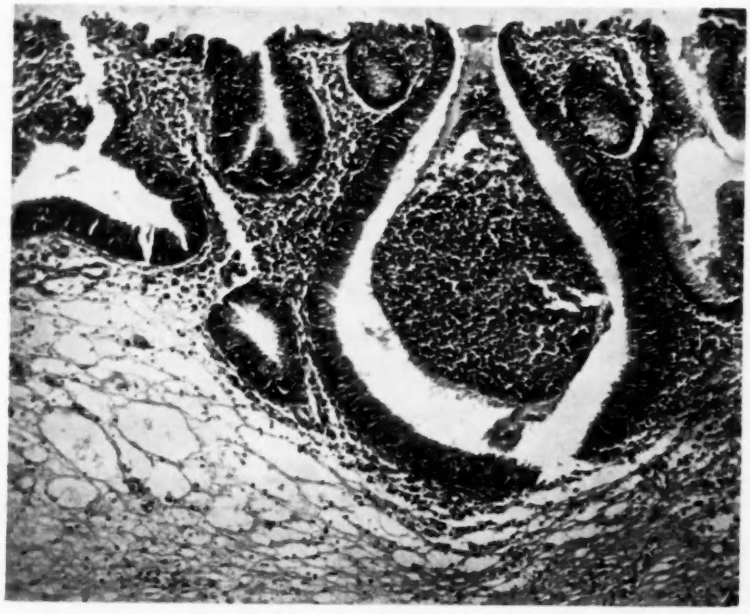

FIG. 11 — (Ash)

The last observation that I wish to show is the development of superficial crypts — pseudo-glands — in some of the cases. Fig. 11 (Neg. No. 68920 — Acc. No. 45767) is from such a case. Female, age 26 years. Bilateral nasal obstruction for one year; frequent frontal headaches; bilateral radical antrum; polypoid mucosa and some pus found. This feature is comparable to the development of Brun's follicles in the bladder and to the cystic cystitis of the urologists. Note the invagination of the mucosa; the mucin activity of the cells, the distention into cysts and the possibility of purulent inclusions. These are not the normal mucosal glands.

In summary, the general pathologist's peep into this special field has impressed him with:

1. The sparsity of suppurative inflammation, i.e., the polymorphonuclear neutrophiles in obviously infected sinuses;

2. The prominence in chronic sinusitis of reticulum in the stroma, especially, and in the basement membrane;

3. The frequency of lymphatic nodules.

4. The development of superficial pseudoglands that may become cysts or abscesses.

I am sorry that I cannot bring to you some practical solution for this very trying problem. I do appreciate the economic importance of the sinuses to you, but I feel that if some way were devised to eliminate them that a lot of people who are now miserable would be very grateful, and would sing your praises in sincere if not sonorous tones.

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DISCUSSION

RALPH A. FENTON, M.D., Portland, Ore.: The Academy again recognizes the great help afforded by Colonel Ash and the Army Medical Museum, in this superb classification and study of pathological sinus tissues.

His analysis of the accumulation of round cells into lymphoid nodules agrees with our experimental findings. Heaping up of round cells seems to occur as a result of diapedesis from small blood vessels. Dean Larsell and I agree that this must be a defensive reaction. And I might say that it occurs to me that these lymphatic glands, resembling tissues which occur in the later stages, might be analogous to the hyperplasia of small lymphoid nodules in the back wall of the pharynx where one can see them. We know from later experiments that the lymphoid tissue, or the lymphoid resembling tissues of the sinus mucosa, drain into the deep glands of the pharynx. We have traced that all the way out, for example, from the frontal sinus by vital staining, and it is quite possible that these lymphoid glands resembling tissues do take up a protective agency and that they do drain out toxic material through the lymphatics which go through the walls of the sinus and out through the ostium and down through the deep pharyngeal glands.

In 1930 I had the temerity to suggest that phagocytic connective tissue cells, so-called histiocytes or reticulo-endothelial cells, were probably present in and contributed largely to the protection of normal sinus membranes and, during inflammation, to the processes of repair. We reported next year, at French Lick, that a few such cells were normally present, arriving in great numbers in acute irritations and accumulating with metamorphosis into fibroblasts during the chronic stage of inflammation. Our work was accomplished by vital staining, and even on such human tissues as we used for experiment in later years was not facilitated by the admirable reticular staining used by Colonel Ash. It is very pleasant to find our experimental conclusions confirmed by his exhaustive study of chronic human inflammations. Incidentally, we could reach no definite opinion regarding the role played by the plasma cells, which also turn up in considerable numbers in such membranes. Has Colonel Ash reached any definite conclusion regarding them?

One hopes that future studies may develop methods for running these reparative methods, let's say, in reverse, and if we can learn how to run them in reverse, we may get rid of edema of accumulated round cells and of these layers of fibrosis, running them backwards, perhaps, but these are problems to which the work of the Army Medical Museum brings material assistance. We should be very grateful to Colonel Ash.

JOSEPH D. HEITGER, M.D., Louisville, Kentucky: Col. Ash has presented an interesting pathological study of the sinus mucosa. Such studies of pathology are important and show us what really occurs within the sinuses. Our therapeutic approach and results must necessarily depend upon the proper interpretation of the pathological processes present. I would recall a quotation from Sir Charles Ballance, of England, which has recently been

popularized by Doctor Henry Williams, of the Mayo Clinic, in his writings, and which epitomizes the importance of pathological study: "Want of agreement among serious workers about a grave question of surgical treatment arises either from the lumping together of clinical conditions having an essentially different pathological basis, or from essential pathological conditions not being well understood. All true and lasting surgical practice is based upon pathology, and when once the pathology of an affection is clearly appreciated, divergence of view as to its treatment ought to disappear."

The pathological process described by the essayist was noticed long ago by Zukerkandl but it attracted little attention. It was made the subject of a study by Doctor Herman Seminov, of Los Angeles, in 1929, while he was at the University of Oregon in the department now headed by Doctor Ralph Fenton. Doctor Seminov has called attention to its presence in his further writings in 1930, 1934 and 1938. He was kind enough to send me a few of his slides which corroborate the findings of the essayist, and a repetition of the details already described will hardly be necessary.

The pathological processes occurring in the nasal sinus mucosa are basic and comparatively simple: exudation, infiltration, proliferation and degeneration. We all know what is meant when someone has been "gyped." By using the initial letter in each of the above-mentioned basic processes one may construct a menmonic which will simplify the idea of sinus pathology. I have found it very useful. One may say that a patient who develops sinus trouble has been "EIPD," the degree depending on the reversibility of the pathological processes present. Exudation, infiltration, and mild grades of proliferation are reversible. Marked degrees of proliferation and degeneration of the sinus mucosa are irreversible and constitute the pathological index of chronic sinus disease which often challenges our best efforts and frequently requires complete selective surgery to fit the individual case and the pathology present.

J. E. Ash, M.D., in closing: I simply want to thank the two gentlemen for their generous discussion. The anatomist, the physiologist and the pathologist have been struggling with the plasma cells for years. They haven't come to any agreement yet, and certainly I am in no position to answer Doctor Fenton's inquiries about their origin, purpose and fate.

RESEARCH REPORT

* * *

STUDIES OF THE OPOSSUM COCHLEA

OLOF LARSELL, Ph.D.*
BY INVITATION
PORTLAND, OREGON

WITH THE cooperation of Doctor Edward McCrady, Jr., of the Wistar Institute, and Doctors E. G. Wever and C. W. Bray, of Princeton University, a litter of 8 pouch young opossums were tested for range of auditory reception at ages ranging from 48 to 77 days after birth. The stimulus intensity in decibels required to produce a response, as well as the range in vibrations per second were measured. The cochleae were then fixed and sectioned for histological study to determine, if possible, the correlation between development of the organ of Corti and the range of response.

The histological study is incomplete because of inability during the past Spring and Summer to obtain additional pouch young of corresponding ages on which to make control studies, by special neuro-histological methods, of development of nerve fibers and neuro-epithelial cells. The several sources from which material was expected were unable to supply it this year, but have promised to do so, if at all possible, during the next breeding season.

On three pouch young, operated by Doctor McCrady in such a way as to injure various parts of the cochlea, and subsequently tested after recovery from the operation, there appears to be a rough correlation between loss of auditory range and the portion of the cochlea which was injured. There appears to be a loss of reception of high notes after injury to the basal coil. This part of the study is also incomplete, requiring more material and control technique, so the conclusions are tentative. It is planned to continue this study as rapidly as material permits.

* University of Oregon Medical School, Portland.

ACCURATE DIFFERENTIAL SECTION OF SENSORY ROOT FOR THE RELIEF OF MAJOR TRIGEMINAL NEURALGIA

Report of Two Hundred Consecutive Successful Cases

ROLAND M. KLEMME, M.D.
BY INVITATION
ST. LOUIS

MY MESSAGE today deals with terrific pain in the face, and the various types of neuralgias that one encounters. I shall try to discuss rather briefly and hurriedly these neuralgias, point out some of the pitfalls, some of the things that should be discouraged, and tell what can be done that will give these patients permanent relief from the *excruciating pain* that they suffer.

The principal neuralgias of the face are sphenopalatine neuralgia, glossopharyngeal neuralgia, trigeminal neuralgia, mandibular joint neuralgia, and others that are of minor import. So that a correct diagnosis can be established these neuralgias must be differentiated from migraine headache; infections of the accessory sinuses; typical jumping toothache; dental caries; infections of traumatic neuritis following extractions; block anesthesia; supernumerary teeth; impacted molars; neoplasms of the alveolar process, jaw and tongue, that is, epuli, carcinoma, etc., and tumors of the Gasserian ganglion.

Trigeminal neuralgia, that in which we are most interested at this presentation, is the one that I wish to discuss in detail. Time will not permit our considering the various other types of neuralgia and conditions mentioned above. I refer the reader to a standard textbook for a complete résumé and method of diagnosis that obtains in any one of those mentioned.

Trigeminal neuralgia, also spoken of as trifacial neuralgia, and by the French as tic douloureux, is a condition that affects the fifth cranial nerve. *This condition is characterized by parox-*

ysmal attacks of lightning-like pain affecting this nerve. The distribution of the pain is more commonly seen in the maxillary and mandibular than in the ophthalmic division, although not infrequently all three branches are involved. The attacks of pain are usually unilateral. About one-half of one per cent of all patients have bilateral trigeminal pain. In this series of cases reported since the introduction of the accurate subtotal resection of the sensory root for relief of major trigeminal neuralgia,[1] fifty-four per cent occurred in women, forty-six per cent in men, sixty-three per cent occurred on the right side and thirty-seven per cent on the left. In none of the cases here reported was the ophthalmic division involved. When the ophthalmic branch is involved, a total resection of the sensory root is done. In two patients, a bilateral accurate subtotal resection was carried out.

The etiological factor of this condition is still unknown. Whether it is due to an infection, a circulatory phenomenon, or some type of physiological upset has not been proven. Infection of the teeth and accessory sinuses as well as exposure to change of climate may be a predisposing cause.

DIAGNOSIS

Major trigeminal neuralgia is the most important form of neuralgia of the cranial nerves because it is the most frequent type with which we meet. All three branches are seldom affected simultaneously, the ophthalmic branch being least frequently involved. The maxillary and mandibular branches are involved most frequently, and in its incipiency, the condition involves either branch about equally. *The pain is in the distribution of the nerve and comes on in extremely severe paroxysms, and as patients express it, like "flashes of lightning." Occasionally when a particular area of a patient's face is touched or stroked, an attack is precipitated.* This area is spoken of as the *"trigger zone."* Curiously enough, many people think that a trigger zone area is essential before a diagnosis can be made. This is not the case. Many patients with trigeminal neuralgia do not have, nor have ever shown signs of a trigger zone. The trigger zone, when present, is most commonly along the vermilion border of the lip and not infrequently on the side of the tongue. The pain of trigeminal neuralgia is probably the most distressing, intractable form of nerve pain. The severity of a major attack cannot be exaggerated.

TREATMENT

After a correct diagnosis is made, the question of proper treatment is to be considered. Treatment of trigeminal neuralgia can be divided into two classifications, palliative and radical. The *use of drugs* such as coal tar products, salicylates, narcotics, and the like *should be thoroughly discouraged* because they are of no benefit. Trichlorethylene is one drug that is sometimes of value. This drug seems to paralyze the sensory fibers of the fifth nerve. The patient inhales trichlorethylene three to four times a day and, if the drug is effectual, will gain relief in about twenty-four hours. This, of course, is only a temporary or palliative method of treatment. Unfortunately, the effectiveness of this drug is so infrequent that its use is very discouraging.

A short résumé of the various types of treatment might be interesting at this time. Trigeminal neuralgia was recognized as early as the middle of the eighteenth century and in 1748 Schlichting introduced peripheral neurectomy for the treatment of this disease. It is to be regretted that this method is still used by many physicians. The results obtained by this type of treatment are comparable to the results following alcohol injection, both of which are palliative. Peripheral neurectomies, in addition to being only palliative, leave undesirable scars. The promiscuous extraction of teeth for the relief of the pain of trigeminal neuralgia should not only be discouraged but thoroughly condemned.

As early as 1756, Andre, of France, described this condition very comprehensively and few additions to his original monograph have been made. It was he who introduced the term "tic douloureux," for painful affections of the face. Fothergill, in 1773, also described in great detail, painful affection of trigeminal neuralgia. The accepted form of treatment up to 1890 was peripheral neurectomies and many reports by Langier, Lizar, Wagner, Mears, and others, can be found in medical journals. In 1891, Sir Victor Horsley carried out an avulsion of the sensory root from the pons. The patient died, which discouraged him from employing this procedure for further treatment. In 1892, Rose of London, and Andrews, of Chicago, introduced the curetting of the Gasserian ganglion. This procedure never became very popular because of the high mortality. In the same year and the following year Hartley and Krause introduced the complete ganglionectomy for the treatment of trigeminal neuralgia. This procedure also was attended with a high mortality.

In 1898, Spiller and Kean in collaboration, suggested the sectioning of the posterior root for the treatment of trigeminal neuralgia, and in 1901,[2,4] Frazier reported the first case of complete section of the sensory root for the treatment of this condition. Since that time the generally accepted and correct operative treatment of major trigeminal neuralgia has been the cutting of the connecting fibers of the sensory root between the Gasserian ganglion and the central nervous system.

In 1906, alcohol injection of the peripheral branches of the trigeminal nerve for the palliative treatment of trigeminal neuralgia was introduced by Levy and Bandowin, and in 1907, Patrick modified the technique and his modification is the accepted procedure for the injection of these nerves. Alcohol injection of the peripheral branches of the trigeminal nerve is a palliative treatment for trigeminal neuralgia which gives relief for a varied period of time, from one month to as long as five years. The average is about eighteen months, after which time the pain recurs.

Since the introduction of the section of the sensory root by Frazier in 1901, various modifications of this procedure have been carried out. The complete anesthesia of the face and the not infrequent eye complication with resultant occasional loss of the eye, led Frazier,[3] in 1915, to introduce subtotal resection of the sensory root, with the preservation of the ophthalmic division. Other modifications ensued. The motor root is now preserved[4] and subtotal resection has been substituted for the total resection in most neurosurgical clinics.

Because of the variations in skin anesthesia following a subtotal resection of the posterior root,[8,9] further refinement in the differential sections seemed necessary. By utilizing the exhaustive studies on the morphology of the Gasserian ganglion, accurate differential section was devised. These studies by Frazier and Whitehead,[5] F. Van Nouhuys,[6] and Davis and Haven,[7] showed that the arrangement of the fibers in the sensory root varies slightly, and, though the fibers occasionally interlace, they generally parallel each other. These anatomical findings, plus the experience of over 200 consecutive cases, show that an accurate subtotal resection can now be obtained, preserving the ophthalmic fibers while yet completely sectioning the fibers of the maxillary and mandibular divisions, thereby preventing the dreaded trophic keratitis associated with total resection or injury to the ophthalmic branch. That this procedure can be carried out accurately is proven

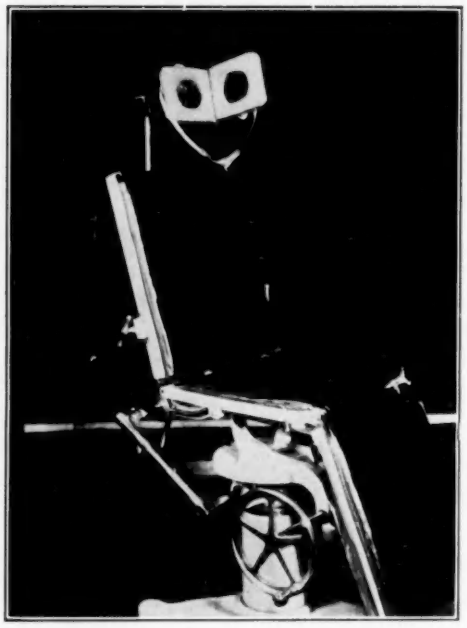

FIG. 1 — (Klemme)

by more than 200 successful consecutive cases. The motor root is preserved. This is essential to the proper functioning of the masseter muscles. The dental profession is far better able to evaluate the importance of the preservation of the motor root than any other group of men. Proper occlusion and the proper fitting of dentures is definitely influenced by the preservation of the fibers leading to the masseter muscles.

The operation is carried out in a sitting position, a simple head rest supporting the patient's head.[1] The usual subtemporal approach is used and the muscles and skin are retracted and held apart with a self-retaining retractor.[1] An opening is made in the bone with a perforator and burr and enlarged to about the size of a quarter. The brain is elevated extradurally and the foramen spinosum exposed. The middle meningeal artery is coagulated at this point and then cut with a knife. The dura propria is stripped from the ganglion and the maxillary, as well as the mandibular branches, are exposed as they leave the ganglion. The ganglion is then injected with ½% novocain and the dura over the ganglion is incised at the junction of the maxillary and ophthalmic divisions. This incision is then extended posteriorly across the ganglion over the

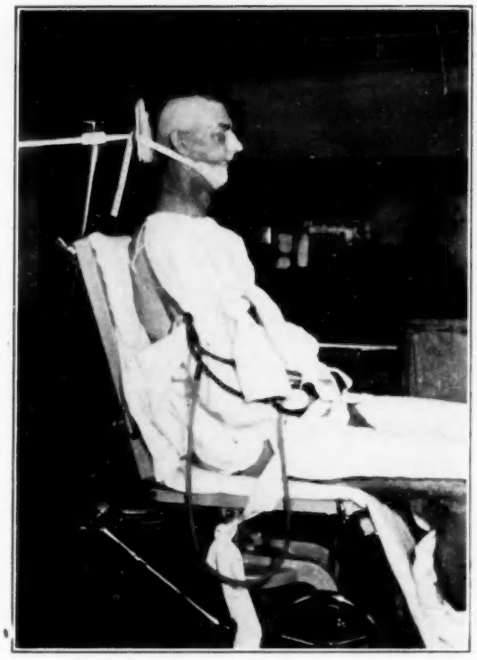

FIG. 2 — (Klemme)

posterior root. The fibers leading to the mandibular and maxillary divisions are separated from the ophthalmic division fibers with a fine spatula. This spatula is gently carried back through the ganglion into the posterior root. A hook is then inserted around the fibers leading to the maxillary and mandibular branches, and these fibers are then cut with a knife or scissors. The motor root lying mesial to the sensory root and running diagonally across the cava Meckelii, is left intact. Ringer's solution is then injected subdurally to belly out the dura, and the wound is closed in definite layers, using silk for the muscle, fascia, galea, and skin.

This procedure has been carried out successfully by me on 200 consecutive cases and since the subtotal resection can be done accurately, I no longer recommend alcohol injections for the treatment of trigeminal neuralgia as a palliative measure. The injection of the nerve is a blind procedure and attended with no little risk. On two occasions, prior to the adoption of the accurate differential section, alcohol injection was done and a complete anesthesia of all three branches was obtained. This definitely jeopardized the eye on the affected side. The risk of the operative procedure which gives permanent relief, is less than on-half of one per cent.

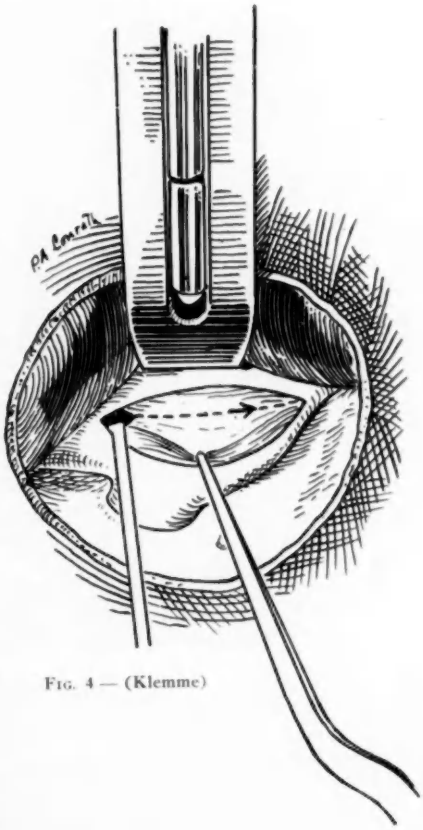

FIG. 4 — (Klemme)

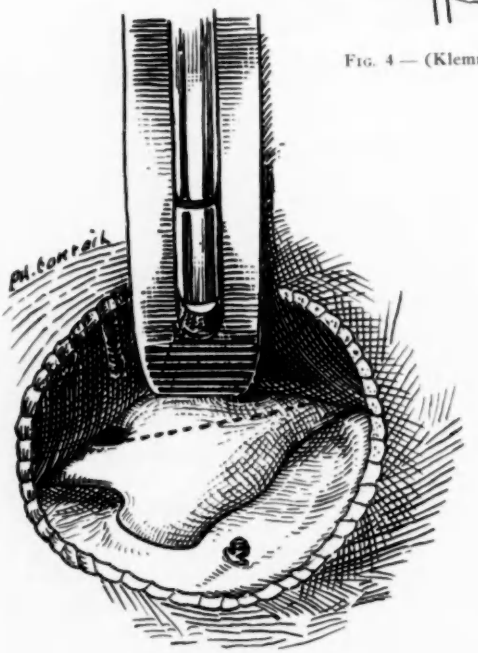

FIG. 3 — (Klemme)

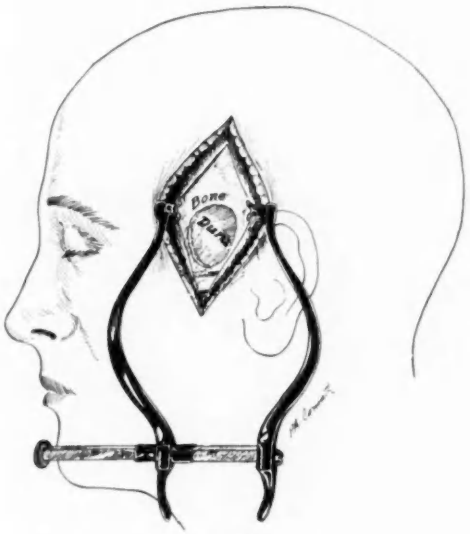

FIG. 5 — (Klemme)

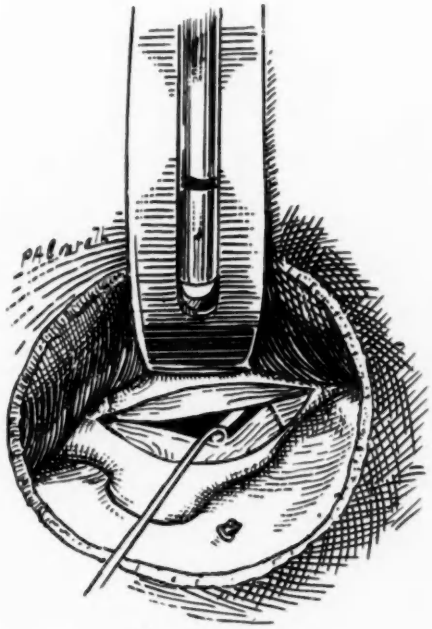

FIG. 6 — (Klemme)

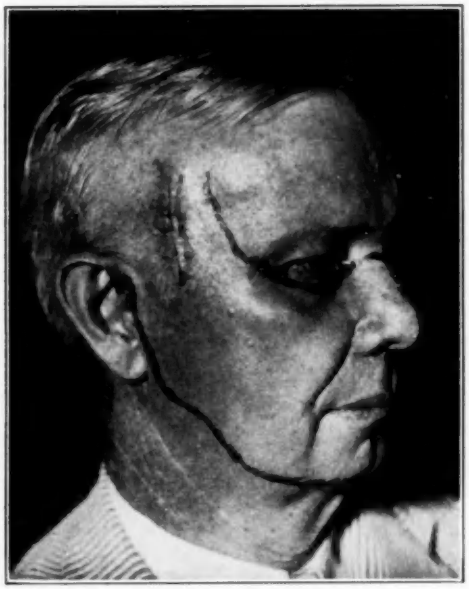

FIG. 7 — (Klemme)

FIG. 8 — (Klemme)

CONCLUSIONS

Accurate differential section, with the preservation of the ophthalmic fibers and the sectioning of the maxillary and mandibular fibers, can be done consistently. Preservation of the ophthalmic fibers eliminates any eye complications such as trophic changes or trauma to the globe of the eye. Preservation of the motor root eliminates any dysfunction of the masseter muscles. The operative risk is less than one-half of one per cent, and the relief obtained is permanent.

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DISCUSSION

HARRY LYMAN, M.D., St. Louis: It is always a pleasure to be present and hear the presentation of something which we feel is a distinct advance in surgical therapeutics. I feel that most of us have been impressed with the fact that this differentiation that Doctor Klemme has described is more accurate than anything that has been done before and is a distinct advance in the control of this terrible affliction.

The members who practice ophthalmology will, of course, realize more strongly than the rest of us even the importance of preserving the oph-

thalmic branch where it is not involved in this pathological process, and the certainty with which Doctor Klemme has shown that, on the screen, must be very impressive to the ophthalmologists.

To those of us who practice ear, nose and throat only besides these features which I have mentioned comes to us the selection of cases. In true tic douloureux the section of the posterior root seems to be an absolute cure, but if the case is not a true tic douloureux, then section of the nerve is followed by partial relief only or in some cases no relief at all. It is in this phase of the question that we are able to contribute our part, that is, in the recognition of other types of pain in the face which may have been described as tic douloureux but which are not true cases of tic. They may be, as he mentioned, sphenopalatine ganglion neuroses. They may be the result of irritations of the nerve by sinus infections, and numerous other causes. By a very careful study of these cases from the ear, nose and throat point of view we are often able to detect factors which would render the posterior root resection a procedure which would not give the expected relief.

Then there is another phase of this question, and I thought that Doctor Klemme would show the slides of actual operations but he did not. However, in the scientific exhibit there are a series of skulls showing this operation in detail, and it brings forcibly to mind the approach to the apex of the petrous pyramid as described and recommended by Doctor Marvin Jones no later than this morning in his course, the approach through the same area that Doctor Klemme utilizes for Gasserian ganglion and using the foramen ovale. The presence of the Gasserian ganglion in that region is a landmark leading directly to the apex of the petrous pyramid in those cases in which it is necessary to attack that locality.

I think if you will look at those pictures in the scientific exhibit you can see that the distance from the outer surface of the skull to the apex of the petrous pyramid is much shorter than you have any idea of unless you have made such anatomical studies.

THE ETHMOIDAL PROBLEM

PROF. ARTHUR DE SA, M.D.
BY INVITATION
RECIFE, BRASIL

WHEN I planned my visit to the United States it never occurred to me that I would have the great honor and rare pleasure of taking part in the present meeting of the American Academy of Ophthalmolgoy and Otolaryngology.

I was on the point of making my last preparations for this second sojourn in your hospitable country when your Program Committee tendered me such a complimentary invitation that I could not but accept it. I saw behind that invitation a homage to Brasil, a republic governed by the same healthy democratic principles, such as yours, and like yours, bent on a good-neighbor policy and desirous of closer cultural relations with her sister republics of the continent. You wished me to take an active part in your discussions and I felt greatly honored in acceding to your wishes. To begin with, I must convey to you — the world teachers of O.R.L. — the most cordial and friendly greetings from the Brasilian Society of O.R.L.

Doctor Ralph Fenton will now read my comments about the ethmoidal problem. I do not propose to bring to your attention anything entirely new but only a modest Brasilian contribution on the subject — a contribution that may perhaps help its solution. This explains, in part, the choice of my thesis, as the contribution to which I refer is represented by a surgical technique, improved by a Brasilian specialist, born in the same state of Brasil as I, and is therefore a contribution from Pernambuco. But that was not the only reason. The subject, among others, has been the object of intensive study by the eminent Prof. George Morrison Coates and, by choosing it, I meant to pay my humble homage to your genteel and noble president. There is nothing that can be added to his excellent work published in 1935 in the Annals of Otol., Rhin. and Laryng. Although four years have elapsed it is still up-to-date and I am happy to say that I am in full accord with his views on the

subject. The anatomical denomination of ethmoidal labyrinth, the region where the morbid alteration have their seat, is very appropriate; the ethmoidal problem itself is a labyrinth. Thus, it is not easy to follow its devious paths or to find our way out by guaranteeing the sufferers of ethmoiditis that which they crave, their radical cure. As the years go by, *surgery is losing ground* in the therapeutic field. The divergence between the preference of the interventionits either for the endo-nasal way or the external via, although each has its own indication, is *expressive* as a *factor* of the *failures* of *cure*. Some *new factors*, so well studied among you in the past years and that only now are receiving attention in Europe, *explain*, to a certain extent, the *cause of failure* of surgical interventions in cases of ethmoiditis. I refer, of course, to the question of dietetics, of bio-chemistry and of allergy. You know better than I that certain congestive phenomena of the ethmoid disappear with an alkaline diet and the ingestion of food rich in vitamins A and D. The writings of Edman on this particular subject, published in the Laryngoscope of 1934, are very interesting. The contribution of bio-chemistry to the study of ethmoiditis and also of other affections of the facial sinuses deserves the closest attention. Stricker, Lewis, Frank and others of your worthy colleagues have published excellent observations showing the value of *biochemistry* to explain the failure of surgical intervention in the ethmoid and other facial sinuses. It is observed, very frequently, a *deficiency* of *calcium* and consequently cases of acute coryza (colds), foul breath, acid urine and the "ph" of 5, 8/4, 6.

We know that there exists an interdependence between the assimilation and retention of calcium and the ingestion of sodium. We therefore must very often advise our ethmoiditis patients to abstain from sodium chloride in their food. These things are generally known in the United States and have been widely divulged in Brasil through Mangabeira Albernax, professor of O.R. in the School of Medicine of Sao Paulo. And what about allergy? Its influence as regards sinusitis is of considerable value. The studies of French Hansel on the subject are notable. Even recently, on May last, he published in the "Laryngoscope" one more study on allergy in oto-rhino-laryngology where he shows the value and repercussion that it may have, not only in affections of the ethmoid but also on the facial sinuses. Hansel is right when he states in reference to the factor — allergy: "The important part played in oto-rhino-laryngology necessitates greater accuracy in diagnosis

and more conservative surgical procedures on the nose and paranasal sinus." I think that the failure of surgical intervention in cases of ethmoditis is due to the fact, more frequently than it is generally thought, of the association of maxillary sinusitis, and also to the circumstance that the specialists do not try to establish, more carefully, this co-participation. In Brasil, my teacher, Eduardo Moraes, professor of oto-rhinolaryngology in the School of Medicine of Bahia, has insistently emphasized the importance of maxillary sinusitis especially in all affections observed around its neighborhood. In the last number of the "Annals of Otolaryngology" that I received — of June, I believe — there is a study of O. E. Van Alyea about the anatomy of the ethmoid. It is a very competent work on the subject which shows the *very close connection between the ethmoid and the maxillary sinus.* The relation of continuity and contiguity are so intimate that it is rare indeed an *ethmoiditis* without the coparticipation of the *maxillary sinus.*

My fellow countryman, Ermiro de Lima, author of the surgical technique that I will be pleased to describe shortly, has observed that in cases of ethmoiditis the maxillary sinus is always affected. In the oto-rhino-laryngological services of the School of Medicine of Recife, Arthur Moura and Edgar Gouveia, my assistants, and I have always found the ethmoiditis involved with maxillary sinusitis, at the same time. In 1932 in Paris, I had the pleasure of attending the annual meeting of the Societé Française d'Oto-rhinolaryngologie. When the paper of Maurice Bouchet and Leroux on ethmoiditis come up for discussion, I heard Higguet, of Brussels, say that in 30 patients of chronic ethmoiditis he found, in all cases, the maxillary sinuses also affected. For that reason he favoured the trans-antral ethmoidectomy because thus, he claimed, he could get "d'une pierre, deux coups." Taking part in the discussion that followed I had occasion to emphasize the necessity of giving the very closest attention not to impair the physiology of the nose in cases of ethmoidectomies. George Coates, in his excellent work on the "Problem of the Ethmoid," is in accord with my point of view and in that particular connection he states: "Although it must never be forgotten that the physiologic function of the nose must be preserved as far as possible, and restoration of, not damage to, this function should be the ideal even if not always obtainable, in any line of treatment advocated."

Well, the great advantage of Ermiro Lima's technique is that he realizes the ideal, in the authorized opinion of Coates, as noth-

ing, from the physiological point of view, is sacrificed. Another great advantage of Lima's technique is that we transform all the recesses of the cavities around the nose into one cavity only, without subsidiary cavities. I think that Lima must have had in mind the same goal of the otologists when they perform a radical mastoid operation for the radical cure of supuration of the ear. The trans-antro ethmoidectomy is not a creation of Lima's. The priority of this technique belongs to Jansen and was divulged by him in 1897 in a congress of oto-rhino-laryngologists in Moscow. Soon afterwards, Winkler, in Germany, discoursed on the same surgical technique. His was very much similar to Jansen's; so much so that Hajek, in his book "Nasal Accessory Sinuses," describing the transantro ethmoidectomy, associates the two names — Jansen-Winkler — to name the process.

In 1898, Goris, in Presse Medicale Belge, refers to that technique. In the following year, Laurent exhibited to the Belgian Society of Oto-Rhino-Laryngology, an anatomical model showing the way into the ethmoid through the maxillary sinus. After him, Bardenheuer, in Germany, and Luc, in France, practiced this process. But this enthusiasm soon passed and the technique was forgotten until 1926 when Horgan published in the Laryngoscope "The Surgical Approach to the Ethmoidal Cells System." In 1929 Turnbull described "An antro-ethmo-sphenoidal operation." In 1935 Pietrantoni published in the Italian Review of Oto-Rhino-Laryngology a study about the trans-antro-ethmoidectomy. After him, in 1936, Futch relates "A Résumé of One Hundred Trans-antro-ethmosphenoid Operations." The observations of Ermiro Lima began in 1930 but he only divulged them in 1936 in the Review of Oto-Rhino-Laryngology of Sao Paulo, Brasil. The access to the ethmoid through the maxillary sinus is rightly preferred in the light of the newest anatomical investigations of Fiol and Terracol, in Europe, and of Van Alyea, among you. The result of these investigations has altered the anatomical notions of the region in question. Also it modified the pathology of ethmoiditis. It is an undisputed fact that the ethmoid has direct inter-communication with the maxillary sinus specially through its antero-external cells. Who knows if the failures of ethmoidectomies in recent years have not been due to the coparticipation of the maxillary sinus, if not always, almost always?

It should be remembered that such infectious coparticipation of the maxillary sinus, in cases of ethmoiditis is nothing new. In

1903 Picqué and Taubert, in their description of the best technique for the treatment of the affections of the surrounding cavities of the nose, mentioned the access to the maxillary sinus as the last phase of the surgical intervention that began by the ethmoid, passed to the sphenoid, then on the frontal sinus and lastly reached the maxillary sinus. Just the opposite of the technique of Jansen-Wimbler, of Turnbull and of that of Ermiro Lima, with the alterations introduced by him. Let us now give a short description of the technique used by Ermiro Lima who is an oto-rhino-laryngologist and also professor of anatomy in the University of Brasil.

ANESTHETICS

We give preference to local anesthetics. Before its application we use an injection of "scophedal." The technique used for anesthetizing is identical to that used in a Caldwell-Luc operation. On the floor of the nose and on the inferior and medium meatus we apply gauze packs moistened with the mixture or solution of Bonain.

OPERATION

We begin by sectioning the anterior antral wall of the maxillary sinus as if we were making a Caldwell-Luc. Then we enlarge the opening in the direction of the nasal process until we have a good view of the anterior superior internal angle of the sinus. It is necessary to be careful in order not to touch the infra-orbital notch. The mucosa is carefully elevated and we proceed to the inspection of the cavity. Bleeding is controlled by suction and the introduction into the antrum of gauze packs moistened with adrenalin solution. Now it is necessary to see the ostium situated around Fiol's oval fosseta. Some times the naso-antral wall shows a kind of convexity making it difficult to inspect the ostium. In such cases we have to make resection of the convexity of the bone, being careful not to injure the mucosa. This resection of the naso-antral convexity was introduced by Doctor Lima. Let us now exenterate the ethmoid through the ostium of the maxillary sinus.

With a Struyken chisel, we enlarge the ostium in a horizontal direction, backwards as far as the posterior internal angle. With a back-biting forceps, of Cordes' type, we have to enlarge the ostium, forward, as far as the superior angle of the nasal wall, just extending to the unciform apophysis. Sometimes, the ostium must be also enlarged downwards, but it is necessary to be very careful not to injure the sphenopalatine artery. After enlarging

the ostium, we inspect the cavity and control the bleeding by suction or gauze packs moistened with adrenalin solution. Then the bulla is visible through a dihedral angle formed by the superior and naso-antral walls. Now we must exenterate the bulla and remove the cells in a vertical direction until we have touched the roof of the ethmoid as it has been established by Doctor Lima. We still scrape out with a curette extending to the nasal bone. Curettage is continued forward in a vertical direction exenterating the ethmoid frontal cells and downwards as far as the "ager nasi." Doctor Lima calls attention to our visibility of the lamina papyracea angle with the roof of the ethmoid — and this is still one of the originalities of his technique. "When both the anterior and posterior cells have been removed the convexities of the anterior wall of the sphenoid come into view.

We are nearly finishing the ethmoid operation. We inspect the cells localized in the angle formed by the nasal bone and ethmoid roof and also the spheno-ethmoidal cells; if they are affected, they must be exenterated. The sphenoid sinus being involved we must "remove the wall flush with the floor of its sinus." To finish, we must make the communication of the maxillary sinus with the nose through the inferior meatus. The operation is over. Packing is not necessary neither suture of the incision of the gengivolabial recess.

POSTOPERATIVE CARE

The antrum cavity is inspected with a nasopharyngoscope after three or four days and afterwards we irrigate with warm saline solution. We often repeat the irrigation two or three days afterwards. After the second irrigation, according to the patient's condition, he leaves the hospital thoroughly healed.

SUMMARY

While for the treatment of otorrhoea there is only one classical intervention the same thing does not occur with the chronic ethmoiditis. In this case we have: the intra-nasal, the external and the trans-antro-maxillar operations. I have the impression that after the new anatomic investigations made about the ethmoid showing its continuity and contiguity with the maxillary sinus, the preference for the trans-antro ethmoidectomy is well justified. Ermiro Lima, originator of the technique described, being a rhinologist and also an anatomist, utilized his anatomical knowledge in his selection of the trans-antro-ethmoidectomy. We have in Brasil, Ermiro Lima, in Rio, Mangabeira Albernaz in Sao Paulo and in

the oto-rhino clinic of the School of Medicine of Recife, one hundred cases each, evidencing that there is always ethmoditis associated with maxillary sinusitis. This morbid association has perhaps caused the failure of the intra-nasal and external ethmoidectomies.

You know better than I how enthusiastically Skillern favoured the surgery of the ethmoid but, in time, his enthusiasm died down due to the innumerable failures of the ethmoidectomies. The famous Skillern was right. We should only propose an operation to those who suffer from chronic ethmoiditis after being quite sure of a successful intervention. All of us who operate ethmoiditis should always have in mind the factor, allergy, so that we may not expose our patients to failure. Before the ethmoidectomy we should remove the obstacles of the nasal respiration since the airation of the ethmoid is an important condition for success. As regards the technique we Brasilians give preference to Lima's method, because it does not injure the physiology of the nose and therefore it realizes the ideal of George Coates' opinion. Lima's method of operation is neither difficult nor dangerous. It does not expose the patient either to meningitis or bleeding. Slight postoperative cares are necessary. As a rule, a week after the operation, the patient can leave the hospital and take up again his daily duties. Those who have submitted to this intervention testify their absolute satisfaction. I hope you will use Lima's method. It is with great satisfaction that I have just finished reading in Charles Imperatori, and Herman Burmans Book, 2nd edition: "Recently this operation has come into great favour because it allows drainage of the sinuses while preserving to the greatest degree the physiologic function of the nose." Before indicating an operation we should remember Delangrenière's words referring to what he calls "The surgical hour," wherein lies the secret of knowing how to fix the operative indications. Reelus said: "Before cutting a human being's skin we should take the knife in our hand seven times." We must, also, remember that an operation should only be performed after our being familiar with the anatomic region. It was not in vain what our Great Master Mosher said, when referring to the exenteration of the ethmoid, that "it was the easiest operation that could cause a patient's death."

DISCUSSION

ROBERT F. RIDPATH, M.D., Philadelphia: The subject of ethmoiditis has always been more or less the bug-bear of us rhinologists. We see a great many patients with ethmoiditis, and at the present time I am trying to write

on this subject for the new publication of Doctor Skillern's original book. I was very much interested in the Doctor's presentation, as it gives more or less of a new method of reaching the disease in the ethmoidal capsule. This is new to me, although the route through the maxillary has, of course, been suggested for sphenoidal infections many years ago, but going through the maxillary with the thought that the Doctor has expressed, that all ethmoidal infections essentially are complicated perhaps by a maxillary infection, if we are sure of this I think the route is justified.

In my experience I cannot entirely corroborate that all ethmoidal infections also are complicated by maxillary or maxillary infections are complicated by an ethmoidal infection. It is true that a great many are, and if they are I do think that perhaps this new route might be thought of.

This subject of ethmoidal infection, as I started out to say, is one on which we have many varied views. When to operate? The operation that we essentially should consider must depend entirely upon the condition in which we find the ethmoidal cells. If you have a suppurative ethmoiditis, with polyposis, I cannot see that this route would entirely be the idealistic manner in which we are to eradicate all of our pathological conditions, but if we have the suppurative ethmoiditis confined to a few cells, then I think, as I mentioned before, it is something for us to consider, especially when we have the maxillary involvement.

RALPH FENTON, M.D., Portland, Oregon: Professor de Sa desires me to say that he associates himself with the ideas expressed by Doctor Ridpath in the main, and he feels that at least in his part of the world when a frank ethmoiditis exists it practically always involves the maxillary. He says, however, when a polyposis exists and protrusion into the nose occurs, naturally they can be much more easily in most cases removed by working in the nose but without sacrifice of turbinates. He does not like to sacrifice any of the intranasal structures at all if that can be avoided. He conceives primarily, as was stated in his paper, that the physiology of the nose should be retained in order to bring about a quick cure.

He asked me to thank the President and the Officers of the Academy for the privilege of making this appearance and to thank you for your earnest attention to what I conceive to be a very brilliant contribution.

I might say, as a measure of discussion on my own part, that on the Pacific Coast Doctor Sewell has occasionally used this approach and we have also used it at the University of Oregon at times. It is known there somewhat as the Sewell approach, but it is a variation, really, from Sewell's approach to the sphenoid, a transcentral approach to the sphenoid which, of course, we have known for a long time, but the original article, which was published not long ago by Doctor Lima will well repay looking up. It has very fine illustrations. I had the pleasure of reviewing it some time ago, I think for the Annals. So I presume it is in our literature.

We are very grateful to Doctor de Sa. I am sure that I express the opinion of the Academy in that regard.

THE MUCUS SHEET ON RESPIRATORY MUCOUS MEMBRANE

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The purpose of this paper is to emphasize the importance of the thin sheet of mucous which covers the mucous membrane of the respiratory tract. Let us confine our consideration to normal mucous membrane and the slight variation which may occur at the start of an acute cold. For several years I have thought that the value of constantly maintaining a mucus covering on the upper respiratory epithelium has been dismissed too lightly, especially in the nose. Supposing the glands which secrete this material should be out of operation for a period of half an hour, we would have sufficient departure from normal to allow any prevaling invader, be it an actually recognized germ or a so-called filterable virus, to pass our first line of defense. This same thought was expressed in a paper which I read before the Indiana Academy in 1934.[1]

We have known now for many years that nasal respiratory mucous membrane must have cilia, actively moving a thin sheet of mucus along, in order to be normally healthy, and that one without the other is, more or less, helpless. For instance, in atrophic rhinitis where the cilia are absent the mucus remains stagnant and soon becomes infected. Conversely, when cilia are removed from their moist surroundings they soon cease to function.[2]

Let us consider a few facts about the cilia which are found abundantly in the nose and nasopharynx but not in the vestibule or ora-pharynx. Their wave like action results in mucus being swept backwards toward the nasopharynx. This movement is not in the form of an unbroken wave but appears periodically in isolated groups of cells. Their action is such that the posterior part of the nose receives a new coating of mucus about every ten or fifteen minutes.[3] The ciliary movement is apparently activated locally and is not dependent upon innervation.[4]

The glands of the nasal epithelium are in two groups: Simple type goblet cells which secrete mucus, appear in the surface, while in the deeper tunica propria are found compound branched glands which secrete either mucus or serum. The amount of mucus normally produced by these glands in twenty-four hours has been estimated as a liter from the two sides. These glands receive vasodilator, vasoconstrictor and secretory fibers from the autonomic nervous system.[5]

So far as the actual chemical composition of the mucus is concerned, we are not particularly interested at the present time. However, the most recent analysis of it still leaves us with the thought that no antiseptic properties are contained in the mucus secretion, except an enzyme called lysozyme the antiseptic action of which is still under dispute.[6]

In studying the histamine content of nasal mucus in relation to polypi and allergy, Buhrmester and Wenner have shown recently that the excessive secretion from the nose even in allergy is not an exudate of blood serum, but definitely a production of the nasal glands.[7] Buhrmester also showed that the viscosity of nasal mucus is decreased by increased temperature and also by the presence of ionizable salts.[8]

We have always thought that vasomotor changes in the nose have been predominant in effecting mucus production. However, Mudd, Goldman and Grant have reported, in a study of "Reaction of Nasal Cavity and Post-Nasal Space to Chilling of the Body Surface," that, "Application of wires within the nasal cavity has usually caused pain and discharge of clear mucus, sometimes also lacrimation and sneezing. The rhinorrhea has occurred both on the side directly irritated and on the opposite side, although on the former more abundantly, and has apparently been little, if at all, affected by the diminished blood supply and shrinking of the mucous membrane incident to chilling the body surface. Discharge from the nose has been at most a rare occurrence in experiments in which the nasal mucosa was not directly irritated." Apparently, the blood supply in the nose does not have as much effect upon mucus production as has been thought.[9]

Aside from the possible action of lysozyme the main function of this thin sheet of mucus is still considered mechanical. In other

words foreign matter and infectious material is caught up by the mucus and rendered inactive until the cilia can sweep it out of the nose through the nasopharynx into the ora-pharynx. Supposing there is no other function of the mucus and it does act mechanically, if the production of it might be suppressed for a short time, as stated before, our first line of defense would be lost.

Since the cilia apparently respond to a local stimulus, since the blood supply of the nose has little effect upon the production of mucus and since the mucous glands are under the influence of the autonomic nervous system, let us consider this system briefly. Of course, there are many influences in the body which indirectly effect the glands such as local irritation, endocrine secretions and the biochemical balance. Their action and relationship may be elaborated later, but for the present the endocrine and biochemical balance have more of a permanent effect than we wish to consider in this paper.

That part of the nervous system which is now called the autonomic has been called by many names in the past, including: Involuntary-1700, Sympathetic-1732, Ganglionic-1764, Vegetative-1800 and finally in 1898 Langly proposed the name Autonomic. The afferent fibers functionally associated with this system are not considered as part of it and this has given rise to the term outflow. Phylogenetically, the automonic system is older than the somatic. There has arisen a feeling that the autonomic system is a highly specialized unstable set of nerves, on the contrary, it is the continuation of the simple nerve arrangement of the single cell organisms and has to do with maintenance of life.

The autonomic nervous system is divided physiologically into two parts called the sympathetic and the parasympathetic. The outflow from the cranio-sacral sections is called the para-sympathetic while those fibers from the thoraco-lumbar region are called the sympathetic. The two sets work in unison, since their two functions are directly opposite in most instances. The two working in harmony maintain the delicate balance which we recognize as the "feeling of well being." Following is a list of characteristics and accomplishments of the two divisions.[10,11]

PARASYMPATHETIC	SYMPATHETIC
Cranio-sacral out flow	Thoracolumbar out flow
Medulated (preganglionic)	Nonmedulated (postganglionic)
Preganglionic fibers leave brain with 3, 7, 9, 10, 11 cranial nerves	Postganglionic from cervical sympathetic ganglion leave with 3, 4, 5, 6, 9, 10, 12
Vasodilators (question at times)	Vasoconstrictors
Anabolic (protective, conservative) on duty during sleep, has much influence on absorption and elimination	Catabolic (emergency and stress) Responds to pain, hemorrhage, shock, fatigue, temperature, emotional strains, prepares for both attack and defense
Contracts pupil, retracts eye ball Slows heart Stimulates flow of saliva Stimulates flow of gastric secretions Stimulates flow of nasal mucus Stimulates G. I. Musculature Produces contraction in Bronchi	Dilates Pupil, protrudes eye ball Accelerates heart Stimulates flow of saliva Suppresses flow of gastric secretions (?) Mostly vasoconstriction in nose Inhibits G. I. Musculature Produces dilation in Bronchi Responsible for "Gooseflesh"
Acted upon exclusively by Atropine, Pilocarpine, Muscarin, Cholin, and Physostigmin	Acted upon exclusively by Epinephrin, Ergotoxin, Cocain

There is considerable latitude possible in the harmonious working before normal limits are overstepped. However, when one or the other side becomes too over-active that normal limits are passed, irregularity results.

In 1909 Eppinger and Hess elaborated the autonomic imbalance in reference to systemic conditions but their work has received considerable criticism because experimental proof is so contradictory. Nevertheless, there seems to be some virtue in the thought which we may apply to our local problem.

The present conception of the origin of a cold is that there must be an exposure in the presence of some invading germ or virus. Since the mucus sheet constitutes the first line of defense let us see if we can put to use some of the apparently isolated facts enumerated. In the case of a normal individual there are many circumstances in life which might constitute an exposure. In a recent paper by Coates, Davis, and Gordon some twenty causes are enumerated and there are probably many others.[12] We live in the presence of contagion all the time but resist the invader many times when it is very virulent but fall a victim to some milder in-

fection at other times. We often see patients who may commit dissipations while on a vacation that in their every day life would constitute an exposure. Probably no more satisfactory explanation can be offered, which would explain why a cold results from an exposure, than that the first line of defense is temporarily inadequate. The earlier writers and teachers began the description of an acute cold with the sneezing and weeping stage. We have learned that there is a period which precedes this in which there is a dry condition of the mucous membrane of the upper respiratory tract. The closer observers have learned to look for the cause of exposure some hours preceding the dry stage.

Let us go back to the chart of autonomic nervous system accomplishments. Here we see the sympathetic division responding to circumstances associated with an exposure, and we have evidence to believe that it is more active than the parasympathetic at these times. As a result there should be vasoconstriction and at least inadequate stimulation of the nasal glands following sympathetic over-activity.

While there is no definite proof of depressor innervation from the sympathetic division to the glands, still when the parasympathetic is inhibited by atropine we are familiar with the profound drying which occurs in the entire respiratory mucous membrane. Although the vasoconstriction which follows may have very little to do with actual gland activity, still it would tend to open the nose and allow air to pass to the naso-pharynx improperly warmed and moistened.[13] Also it would allow air to pass over the mucus without sufficient contact to allow the foreign matter and infected material to become entangled.

If this paper stimulates more thought and consideration of the formation and maintenance of nasal mucus, for which we have no substitute, the writer will be more than happy.

SUMMARY

1. Normal respiratory mucous membrane especially nasal, needs active cilia moving a sheet of mucus to be healthy.
2. Perfectly normal, healthy nasal mucosa at times will resist the most virulent invader.
3. Inhibiting the production of mucus would constitute an exposure.

4. Secretory parasympathetic innervation to the nasal mucous glands has been reported.
5. A predominant activity of the sympathetic is found to coincide with circumstances associated with the common exposures to cold.

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DISCUSSION

A. C. Hilding, M.D., Duluth, Minn.: I was very happy to read this paper of Doctor Leasure's. I have long felt that the mucus sheet which covers the respiratory surfaces is of utmost importance and perhaps the central physiologic factor there. What little we know about it should be thoroughly grasped by every rhinologist and I think that every effort should be made to learn more about it. That it has an important mechanical function is now well established. Our surgical and office treatment is likely to be doomed to failure unless we take these mechanical factors into consideration.

In one of our instruction hours during this meeting we studied the various results of interfering with the ciliary mechanism surgically in the

sinuses of animals. It certainly has a tremendous importance and it is a mechanical thing which must be understood. However, I am personally thoroughly convinced that the mucus sheet has other important functions besides merely the mechanical ones. Doctor Leasure has indicated that the lysozyme, the enzyme in there which destroys bacteria, is still under dispute. I do not see how its antiseptic properties can be under dispute. It is a powerful antiseptic, one of the most powerful that I know anything about, and one can actually watch the lysozyme destroy bacteria if one puts some susceptible ones under a microscope and subjects them to the action of lysozyme. In general, they act in two ways. Watch a single germ, a single coccus, and it will begin to swell up until it reaches a certain size and then simply burst and actually fragment like a bursting shell. Others, on the other hand, swell and grow fainter and fainter in outline until they must disappear like a whisp of smoke.

The objection is raised that this chemical action is directed only against non-pathogenic bacteria and not pathogenic ones and, therefore, it is not of any importance. I think that matter is still open for discussion. How do we know that these so-called non-pathogenic bacteria would not be very pathogenic if we did not have lysozyme not only in the secretions of the respiratory tract but all through the whole body, in all of our tissues and practically all of our fluids.

Doctor Leasure has called attention to the neurologic controls of glands and gland activity in the nose. I think he went to a considerable effort to gather this material, and it seems to me there lies a tremendous field of paramount importance awaiting some venturesome research man. I do hope that Doctor Leasure goes on with this study. What do we know about the nervous control of the glands in the nose? What do we know about their chemical control or how the secretion is produced — even the simple matter of mucin. It is said there must be about 2 per cent mucin to maintain the proper viscosity. How is that 2 per cent controlled and how is it altered as more or less is needed? We know nothing about these things; in fact, chemistry in general is very little understood.

Then this other mechanism, the connection of the external skin of the body, especially lower extremities, with the interior of the nose. Our grandmothers knew there was such a connection. We feel it every time there is a draft or we have wet feet, but what is the significance of it, what is the mechanism, and what is the normal relation between the skin and the interior of the nose?

Doctor Leasure has opened up a tremendous field and I want to repeat, I do hope that he follows it because we need much information.

J. K. Leasure, M.D., in closing: I have nothing more to add except to thank the audience for their kind patience and Doctor Hilding for his discussion. Since both branches of the autonomic nervous system stimulate the gastric saliva, why should one's mouth dry out?

AN OPERATION FOR THE RELIEF OF CONDUCTIVE DEAFNESS

O. Jason Dixon, M.D.
KANSAS CITY, MO.

I know of no better way of describing the origin of this operation for the relief of conductive deafness than to quote from one of the addresses of Sir Thomas Lewis in his discussion of the application of clinical medicine when he says, "Knowledge that is to be applied usefully to the health of mankind will almost always come by a series of steps, the first of which is the recognition of the human need, the last of which is the application of a test directly to the human problem. It is in the nature of things, however many steps may intervene, that the first and last must be clinical: as it is also in the nature of things that almost all important physiological discoveries that are immediately applicable to the treatment of disease have their original source in clinical observation."[1]

My first thought that some method might be developed for the restoration of middle ear deafness occurred to me about fifteen years ago when I observed a sudden restoration in hearing in a young man upon whom I had just done a bilateral mastoidectomy with open wound drainage. He had been totally deaf for several years and came to me only for the relief of the suppuration of the mastoid process. It had never occured to him that he might hear the spoken voice again, and he was even more startled at the result than I. My anxiety over the ultimate result somewhat dampened my enthusiasm. This, however, he and his relatives took as a form of professional modesty.

As the postauricular wounds healed, hearing diminished, and when he left my care with both wounds tightly closed he was practically as deaf as ever. This operation, together with other observations, such as the removal of adenoid tissue about the mouth of the Eustachian tube in children, and the repeated temporary relief of the old Politzer bag inflations, impressed upon my mind the firm conviction that something could be done permanently and completely to ventilate the middle ear which would permit the en-

trance of the atmospheric pressure and serve as a substitute for the normal, functioning Eustachian tube.

It was not, however, until several years later that I carried out an extensive series of experiments on the dog, and particularly in studying wound repair, that I devised the method of tightly closing the mastoid wound and the replacement of the periosteum and soft tissue flap.[2]

From this operation it was only a short step to the extension of the wound downward upon the neck and the fashioning of a full thickness skin lined tube for the entrance of air to permit ventilation of the middle ear.

In 1934, a young banker, Mr. K., came to me and under local anesthesia I opened into his antrum through the mastoid process. Prior to operation he had been totally deaf to the spoken voice. Immediately after the operation he could hear a whisper. His hyperacousis became so annoying that he left the city to escape these normal noises.

Returning to the bank, he wrote me a most encouraging letter stating that his chief difficulty was to keep people from shouting at him, and that he could no longer endure the sound of the adding machines in the back offices where he had been recently assigned. I had sent him home with a fistulous tract leading from the mastoid antrum to the outside skin which was lined only with granulation tissue. A rubber tube had been inserted down to the antrum, about which I had hoped that scar tissue might form, leaving a patent channel. The normal healing processes gradually closed this fistula and the patient was again deaf.

In order to produce a permanent opening for the entrance of air into the mastoid antrum, I decided that a skin lined tube taken from the neck would be necessary, and that it would have to be fixed within a bone channel made through the mastoid process, permanently ventilating the middle ear. After describing this to my friend Doctor Earl Padgett, and explaining how this patient had reacted, a full thickness skin lined tube was fashioned from the postauricular skin extending down and back upon the neck, leaving the raw surface on the outside in apposition with the freely dissected mastoid cells.

SELECTION OF PATIENTS

Patients with conductive deafness respond best to this procedure, although I have had some marked success with several

patients with variable degrees of nerve deafness. Extreme care should be taken in their selection, since the operation is not an emergency.

The patency of the Eustachian tubes should be tested, although the fact that patients do not have patent Eustachian tubes does not mean that they are not eligible for this operation, because I have had some of my most startling results with patients who had never been conscious of any aeriation. The restoration of the ventilation of the middle ear and the return of the physiological action of the Eustachian tube is dependent upon the entrance of air through the new tube which I form.

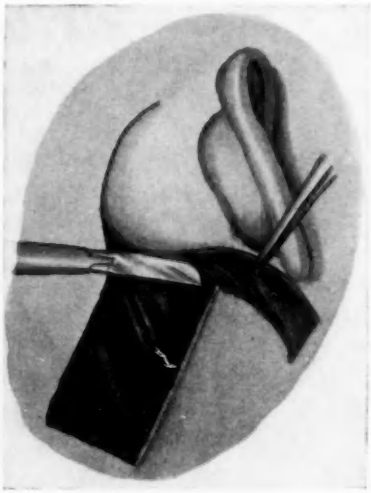


FIG. 1 — (Dixon) — Primary incision and dissection of neck flap.

SURGICAL PROCEDURE

The primary incision should extend deeply through the periosteum and down to the bone and be placed back far enough to follow the posterior border of the external ear as it is compressed against the head. The tip end of the incision extends to about the junction of the lower third of the mastoid process at which point it leaves the bone and is just deep enough to pass completely through the skin. This skin flap, measuring from the mastoid process to its lower tip, is about 5.08 cm. long by 1.9 cm. wide. (Fig. 1.) The pattern of this incision is first outlined with colored

merthiolate which is applied with a small wooden applicator, and the size depends, of course, upon the size and shape of the mastoid process. The Roentgen studies help in determining this. It must be kept in mind that the flap cannot be enlarged, but that it can always be reduced in size.

After this flap is cut and dissected, the postauricular incision is made through the periosteum to the bone. (Fig. 2.) The cortex

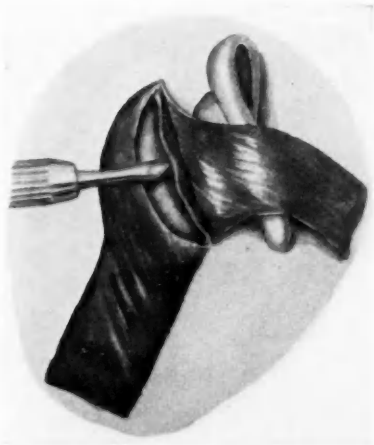


FIG. 2 — (Dixon) — Postauricular incision carried through to the bone, and exposure of the cortex.

is exposed in the usual manner and the antrum freely opened with a sloping of the bone margins and smoothed out with all angles pointing to the antrum. It is not necessary, in fact inadvisable, to open the lower third of the mastoid process, as this destroys the bony support and delays healing. It is important to have a good opening into the antrum, with no sharp projecting angles and no free pieces of bone.

The skin flap is then sutured with the lower surface out, with the stitches tied so that the knots go into the canal where they may slough out. (Fig. 3.) These interrupted sutures should be close enough together to approximate the skin borders smoothly and snugly. (Fig. 4.) The tubular graft is held rigid while being fashioned, by the use of a tension suture. The end of the tube is folded back upon itself and sutured so that all raw edges within the skin

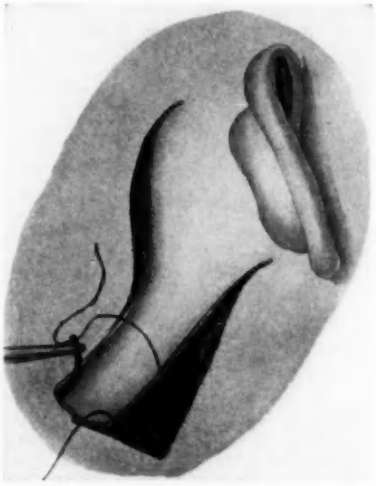


FIG. 3 — (Dixon) — Suturing of the skin flap with the knots tied on the inner side, where they will slough out.

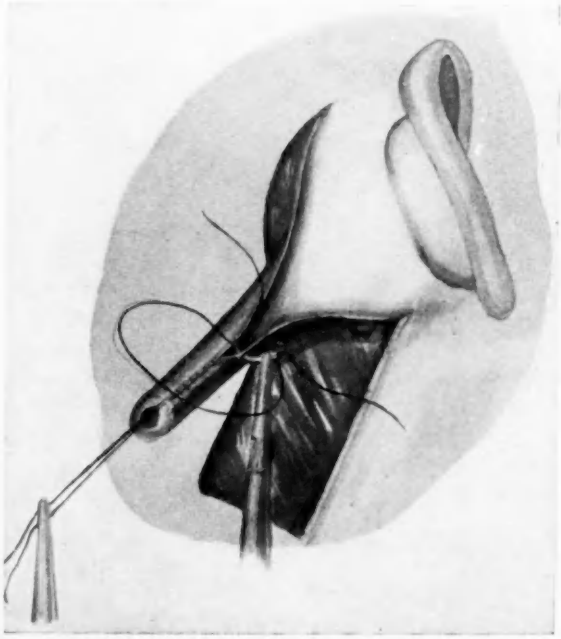


FIG. 4 — (Dixon) — The borders of the tube approximated smoothly and snugly. The tube is held rigid during this procedure by the use of a tension suture.

FIGS. 5 and 6 — (Dixon) — Rubber catheter inserted. The edge of the tubular graft is folded back upon itself and sutured so that all raw edges within the skin lined tube are eliminated.

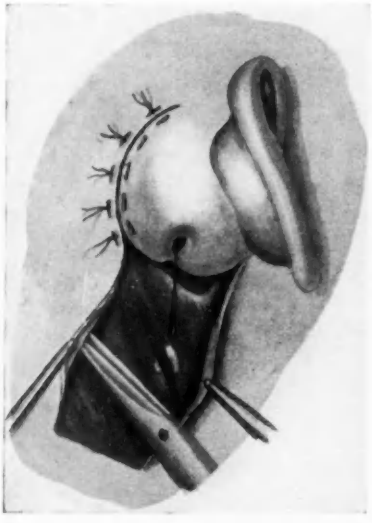


FIG. 7 — (Dixon) — The neck wound is undermined about its entire border approximately one inch.

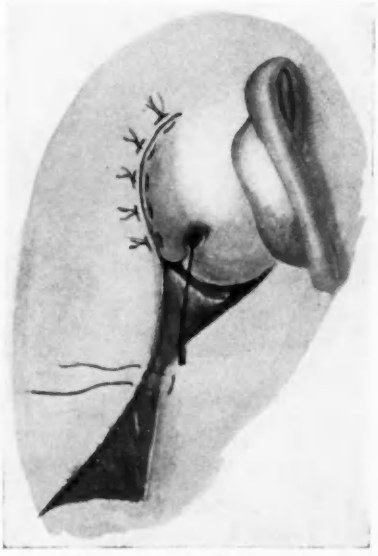


FIG. 8 — (Dixon) — The postauricular wound is sutured after insertion of tube into the antrum, and the neck wound is sewed down to the square tip.

lined tube are eliminated. (Figs. 5 and 6.) The neck wound is undermined about its entire border approximately one inch. (Fig. 7.)

There are two areas where a bit of trick surgery is applied. One is at the lower angle of the wound where I find it quite sufficient to sew down to the square tip (Fig. 8) and pick up this triangular excess of skin and with scissors cut it entirely away and discard it. (Fig. 9.)

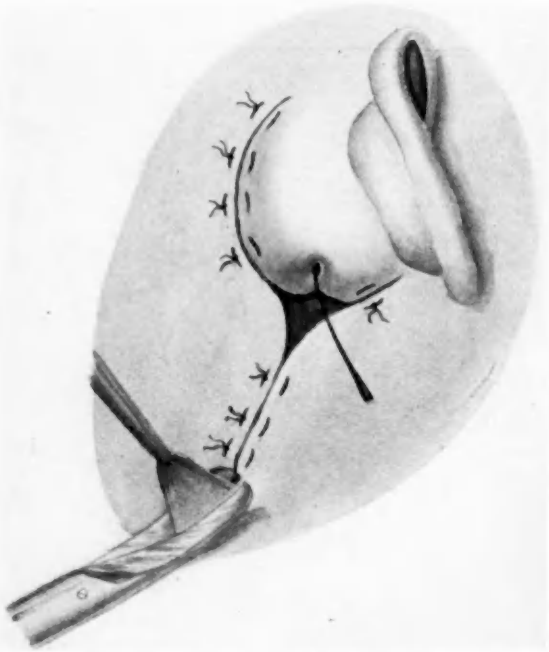


FIG. 9 — (Dixon) — The triangular excess of skin is cut away and discarded.

The next suture is the most important of all. This is the triangular surface exposed just beneath the external orifice of the newly inserted tube. It is important to exercise meticulous care in approximating all skin borders without any unnecessary tension. I use a double continuous purse string suture of triple "ooo" absorbable pyoktanin which when snugly tied leaves no raw surfaces. (Fig. 10.) Just prior to this final closure, the newly placed flap should be lightly compressed to remove all blood clots beneath it. It is especially important that the skin lined tube when primarily

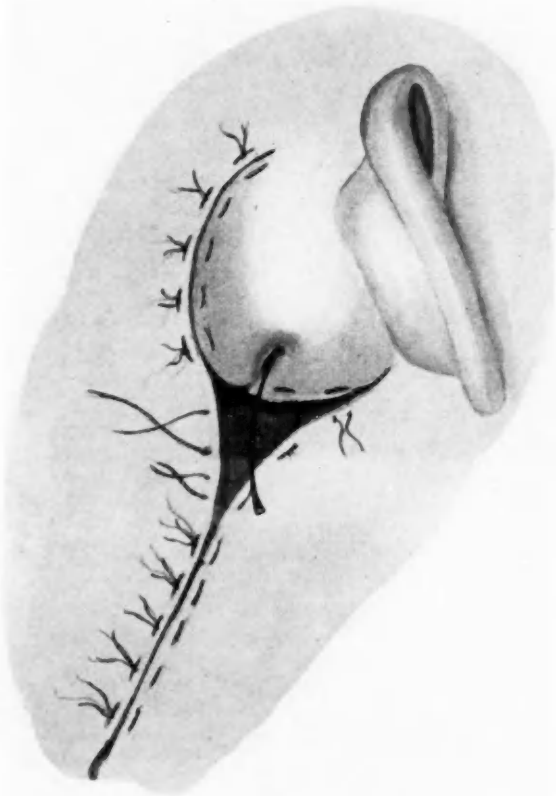
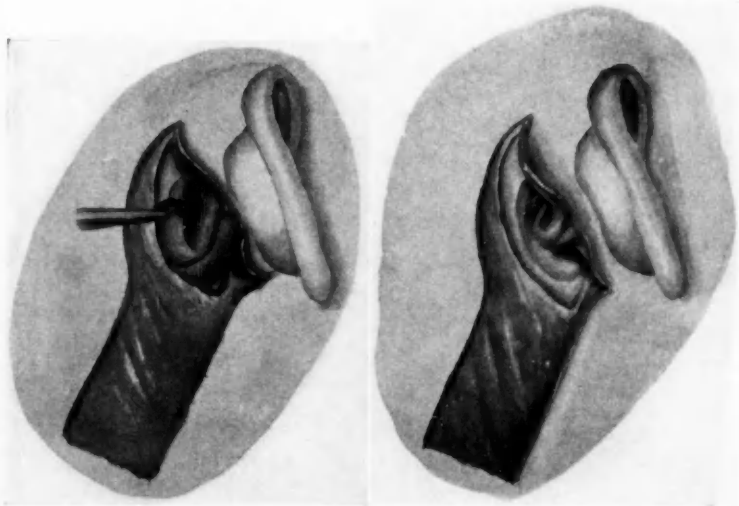


FIG. 10 — (Dixon) — The edges of the triangular raw surface beneath the external orifice of the inserted tube approximated by the use of a continuous purse string suture.

inserted (Figs. 11 and 12) should not again be disturbed, as wound healing begins instantly and it is greatly retarded by repeated trauma. The number eight male rubber catheter (Fig. 13) which extends from the external opening is now inserted and trimmed off level with the skin.

The entire wound is now painted with collodion (Fig. 13) and a pliable parrasine mesh is placed directly over it with lateral reinforcement of an extra layer running transversely over the neck wound. (Fig. 14.) This is held in place by applying a coat of collodion which is quickly dried by fanning during its application. This mesh keeps the wound clean and sterile and because of its



FIGS. 11 and 12 — (Dixon) — Insertion of the tubular graft into the antrum.

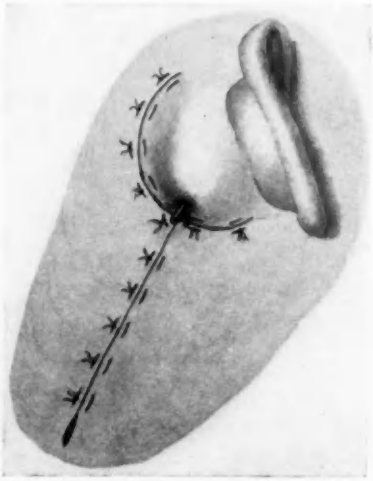


FIG. 13 — (Dixon) — The number eight male catheter extending from the external opening. The entire wound completely sutured and painted with collodion.

flexibility premits normal movements of the neck without separation of the wound.

Soft marine sponges, which are sterilized by a special method, are placed over this postauricular area and the usual mastoid bandage is applied.

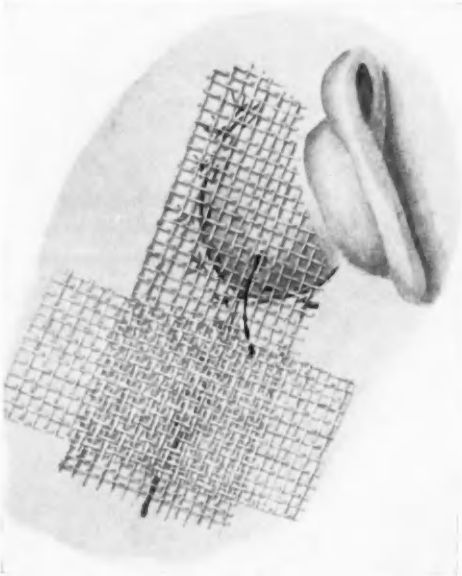


FIG. 14 — (Dixon) — A pliable parrasine mesh placed directly over the postauricular area with lateral reinforcement of an extra layer running transversely over the neck wound.

Only the outer dressing is changed each day, while the postauricular dressing is left intact for about one week, at the end of which time it is removed bringing the remaining sutures with it and the wound is usually healed by primary intention. The marine sponges are moistened each day with sterile saline solution applied directly to the sponge with an eye dropper. This adds to the comfort of the patient and irritation to the external ear is avoided.

The patient is usually up on the second postoperative day, and there is practically no postoperative discomfort.

WOUND HEALING

Experiences in experimental and clinical vascular operations[3] have impressed upon me the importance of the single primary ap-

plication in wound repair. The raw area of this skin flap should be harmed as little as possible and should be folded back upon itself with the raw surfaces in apposition while the bone surgery is being completed. It must also be kept warm and moist while the tube is being fashioned.

Fortunately, as the skin dissection is carried downward upon the neck, it becomes thinner unless too much subcutaneous fat is included. I usually trim this end of the flap so that when it is folded back upon itself it is no thicker than the remainder of the tube. This helps to form a natural graft so that a better connection can be made with the opened antrum.

I find that in cleansing the mastoid wound, and particularly in removing blood clot remnants from the antrum, that it is better to use soft marine sponges that are kept moistened in warm sterile saline solution. It is also an advantage to pack the antrum with a small piece of sponge as soon as it is opened and then complete the surrounding bone work. When this small sponge is removed, it will be found to be covered with small spicules of bone which would have fallen into the open antrum, and it is doubtful if they could have been removed completely.

The physiology of wound healing is no different about the ear than any other tissue within the body. However, some of the fundamentals in the structural changes which have been created must be kept in mind. For example, the periosteal bone covering has been removed and within the bone tube underlying raw surfaces of the full thickness of the skin graft have been placed directly in apposition against this bone. There is nothing particularly complicated in this wound repair. In small areas where the periosteum is not replaced, scar tissue merely forms as a normal process of wound repair.

There is very little attempt on the part of nature to replace this cancellous bone which has been removed. In the depth of the wound which consists of a bony space or cavity, which, having been constructed as a cavity, maintains that tendency to remain so, and we therefore need have no concern of bone replacement and obliteration of the cavity. However, my experience with this type of clinical problem teaches me that the larger this cavity, the better is the restoration and the more permanent the hearing.

Nothing enlarges this cavity and maintains this space about the antrum so well as previous bone disease. This is especially true where there has been a bony cavity formed and filled with soft

caseous debris in which the granulations have sloughed off with no tendency to return. The bone scar consists of hard, smooth eburnized bone with practically no nutritive supply. This cavity, having once been so well formed, continues to maintain this fixed size and shape.

When the end of the skin tube is placed into this cavity or connects directly with it, it is only a matter of ten days or two weeks until an extension of granulomatous buds form about the surrounding skin tube and adjacent newly formed air channel.

Obviously, this temporarily interrupts ventilation and retards the restoration of hearing. Within a few weeks, however, the normal process of wound repair, with sloughing and scar tissue contractions permanently reopen this pathway for the passage of air and hearing is again restored. Seldom does this granulation debris leave behind any permanent damage to the auditory mechanism.

Due to the fact that this newly placed tissue is directly connected with the nasopharynx by way of the old Eustachian tube, it is to be expected that some infection would be present.

Usually a narrow strip of hair is included in the flap on the posterior border near the end of the tube down along the back of the neck. This hair grows, and in the course of a few weeks, finds its way to the outer opening and acts as a permanent and continuous drain. A few patients have a curling or massing of this hair about the inner end of the tube. This, I find, is easily drawn outward by the use of a number six steel crochet hook, and the hair can be trimmed or allowed to grow and fit in with the rest of the hair.

SUMMARY (FIG. 16)

This operation is a combination of soft tissue plastic surgery and surgery of the mastoid process. Of the two, bone surgery is much more important as it involves vital areas. Any otologist who is familiar with mastoid surgery should have no difficulty in performing this part of the operation. The construction of the plastic skin graft requires meticulous care, but it is not especially difficult.

The operation consumes about one hour. The hazards are practically limited to those of a general anesthetic. In none of my patients have I at any time had any complications which endangered the life of the patient, or left him with any handicap, nor does it impair the hearing which may be present.

In none of my patients have I yet failed to secure enough improvement in hearing and relief in tinnitus, that did not warrant operation. A sufficient number of patients have been operated upon to warrant the conclusion that once the tubular channel is formed, it remains permanently open. Entirely unanticipated, tinnitus has been interrupted in every one of these patients. Only where there is a temporary or intermittant blocking of the tube has there been any recurrance. A more complete study is being made of this phase of the procedure and will be presented in a separate paper.

The success depends first upon the proper selection of patients, second upon the meticulous care in the operative technique, and third upon the regular postoperative care until the wound is entirely healed.

SUMMARY (FIG. 15)

1.	Bilateral deafness	100%	
2.	Marked impairment, unilateral	11%	
3.	Progress, worse	100%	
4.	Known cause	44%	
5.	Known draining ear	19%	
6.	Longest duration	43	Years
7.	Shortest duration	1½	Years
8.	Average duration	13	Years
9.	Oldest patient	70	Years
10.	Youngest patient	4	Years
11.	Had worn hearing aid	36%	
12.	Male patients	50%	
13.	Female patients	50%	
14.	Quinine taken over a period of years	.5%	
15.	Bone conduction increased	100%	
16.	Air conduction decreased	100%	
17.	Nerve impairment	11%	
18.	Tinnitus	97%	
19.	Headache	50%	
20.	Colds	67%	
21.	Dizziness	19%	
22.	Immediate restoration of hearing	61%	
23.	Restoration of hearing for the spoken voice:		
	0-50% — 29%		
	50-95% — 71%		

24. All patients have restoration for sound.
25. All but one of the best results had immediate restoration.
26. All but two of the best results had a loss of hearing after regaining following operation, and then had a permanent restoration.
27. Improvement, marked, in unoperated ear.... 35%

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DISCUSSION

SAM ROBERTS, M.D., Kansas City, Mo.: I am sure that all those presenting papers on the surgery of deafness are serving a good purpose. They should be congratulated and should be encouraged to continue. None of them feel that he has a finished product, in fact all feel there is more to do and their work should be inspirational to the rest of us to contribute what we can.

I have worked along with Doctor Dixon to a somewhat limited degree, but I have seen six of the patients whom he demonstrated one night at the University of Kansas. All of these patients explained that they heard much better and were happy with the operation, some of them at that time wanting to go through the second ear. Nearly all of them, as I recall, were bilateral that Doctor Dixon showed at the University.

I think that from a scientific standpoint Doctor Dixon ought to have audiograms before and after, or at least some form of functional testing. None of the patients that I have seen complained of the result except one. This one patient happened to be my own on whom he had operated and whom I had diagnosed as otosclerosis. He had very patent tubes. That is why I do not see why a supernumerary tube would help him, but nevertheless Doctor Dixon stopped his tinnitis, which I could not do. His hearing remained the same.

I hope that in three years from now, or two years from now, these gentlemen will come back before the Academy with their final results. We do not know in our fistulization operation whether the fistula is going to remain open. We do not know what the percentage of fatalities is going to be. Certainly, there are likely to be some fatalities. I know that I would be

very much alarmed in doing a radical if I knocked off part of the semicircular canal. I do not know that Doctor Dixon's operation is anywhere near as dangerous a procedure.

As I told him in the beginning, it makes no sense whatever to me. The answer is not a pathological condition and it does not appear as a physiological process, except that we are not able to open the tubes otherwise. I do not mean by simple inflation, but I do mean by the work of Costen of St. Louis where he builds up the posterior bite and those tubes open up and remain open and the patient's hearing is better. But I am sure that we all congratulate them and want to encourage them to do further work.

J. B. COSTEN, M.D., St. Louis, Mo.: When I had the privilege of examining Doctor Dixon's cases a short while ago in Kansas City the main thing that interested me, of course, was the probable physical condition of the nasopharynx and the pressure condition in the nasopharynx that made such a change of benefit by whatever the effect is on the eustachian tube. It occurred to me that if there was sufficient nasal obstruction to give the well-known slightly negative pressure in the nasopharynx, the nasal part would be corrected as we all know. It also occurred to me that if there was infection pouring from the sinuses to affect the eustachian tube or over-closure of the jaw to cause its inadequate function, this could also be corrected. So in the cases that I saw I was very much interested to look for the probable reason the tubes in those cases were not patent or intermittently were not so. It appears to me that something is happening in the tube of that patient in whom the levator palati muscles are tense or the levator palati muscles are not functioning, so that as soon as an adequate balancing chamber for air pressure is established, the result is obtained.

I wish to testify that the result really is obtained, because I was skeptical and am a good enough friend of Doctor Dixon that I would not hesitate to tell him that he was off if he were sour. I think that instead of taking away the drum what has been accomplished is probably some kind of a reserve chamber which helps to equalize the pressure in the nasopharynx.

An interesting thing about a few of the cases is that on the operative side there is improvement and then after a few months on the non-operative side the eustachian tube is found to be normally patent and in any examination is not found to be closed. I can only surmise what is happening there, but when relaxation of the spasm, or whatever abnormal palate reflex is occurring to close the tube, is relieved on one side perhaps it happens on the other side. That is also said on the basis of fact, because the opposite tube does actually change in its patency.

H. G. KOBRAK, M.D., Chicago, Ill.: Doctor Dixon has treated his very interesting problem mainly from the surgical standpoint. He seems to explain the improved hearing after making the fistula as due to improved ventilation of the Middle Ear. There is, however, another physical principle which could be the explanation for the phenomenon. This principle is the "Direct application of sound into the cavity of the Middle Ear."

At the University of Chicago, experiments have been performed together with Doctor J. R. Lindsay and Doctor H. B. Perlman, in which the application of sound through a fistula into the Tympanic Cavity was studied. These investigations were carried out in the animal, on human cadaver specimens and on patients. Experiments on fresh human temporal bones show that airpressure changes in the Outer Ear and in the Middle Ear produce movements of the labyrinthine fluid. The experimental procedure is demonstrated in Fig. 1 (left half). The result was striking in that changes in pressure in the Middle Ear produced a far greater movement of labyrinthine fluid than identical changes in the Outer Ear.

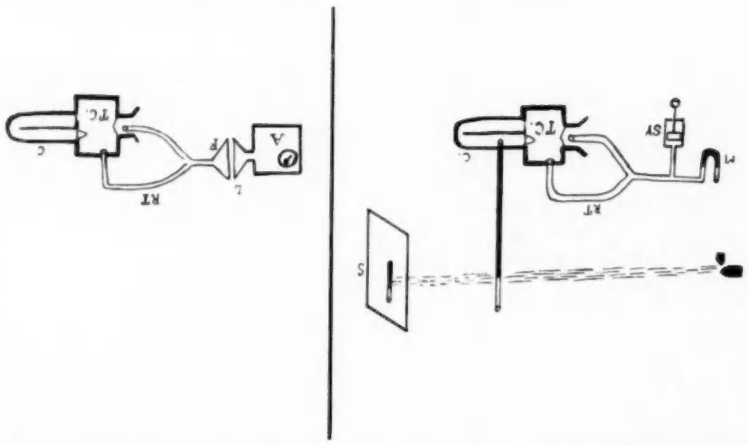

FIG. 1 — (Kobrak) — Diagram illustrating the method. The left half of the figure demonstrates the arrangement for static experiments on human cadaver specimens. The airpressure changes are produced by the syringe (SY) measured in the manometer (M) and conducted by rubber tubes (RT), either into the outer ear or into the tympanic cavity (TC). The meniscus of the capillary inserted into the cochlea (C) is visualized on the screen (S).

The right side shows the experimental procedure for acoustic stimulation in the rabbit. The stimuli are produced in the audiometer (A). A glass funnel attached to the loudspeaker (L) leads the sound energy either into the outer ear or into the tympanic cavity (TC). The effect on the cochlea (C) is measured by observing the tensor tympani reflex of the opposite ear.

Conditions which resemble the mastoid fistula in the human can be produced in rabbits by making an opening into the Bulla Ossea. The right half of slide 1 shows the experimental set up. The acoustic stimulation of the cochlea is measurable by observing the Middle Ear Muscle Reflex. This reflex and its utilization as indicator of cochlear function in the animal has been carefully studied previously. (Value of the Reflex Contraction of the Muscles of the Middle Ear. Arch of Otolaryngology 21; 39, 1935.) It can be demonstrated in the rabbit that the threshold for sound applied into the Middle Ear cavity is considerably lower than the threshold for sound stimuli applied into the Outer Acoustic Meatus. Slide No. 2 shows this condition. It is evident, therefore, that the cochlea can be stimulated successfully through the air of the tympanic cavity. This pathway of sound conduction, which has been rather neglected in the literature, has been named aerocochlear sound conduction.

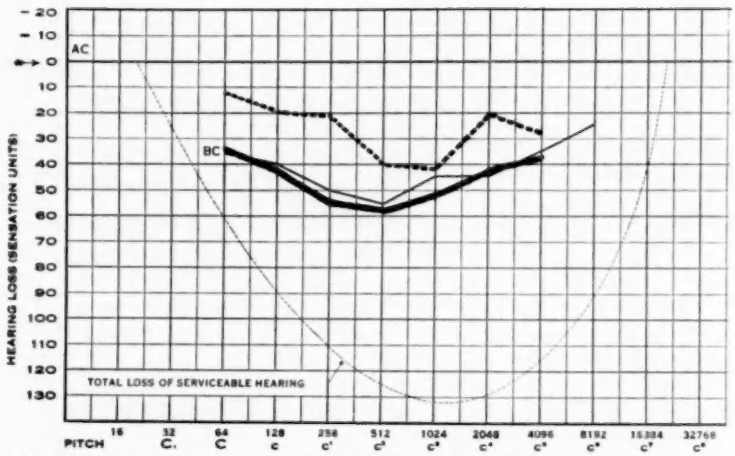

FIG. 2 — (Kobrak) — Chart showing the thresholds of the tensor reflex of the rabbit for sound carried directly into the outer acoustic meatus (indicated by solid line) and for sound carried directly into the middle ear. (Indicated by a dotted line.) The "hearing gain" is obtained by application of sound to the middle ear.

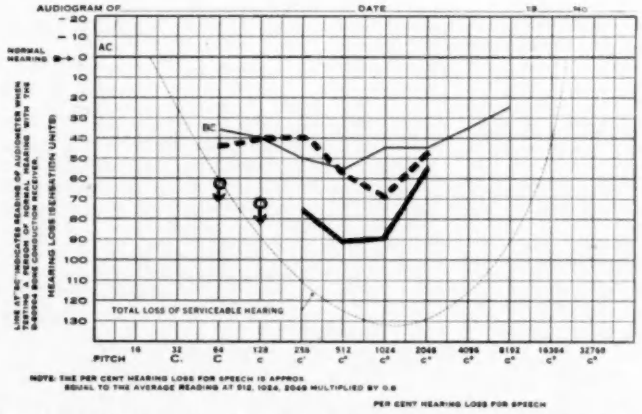

FIG. 3 — (Kobrak) — Function of the cochlea of a rabbit, with destruction of apical coil. The solid line indicates the function by application of sound into the outer ear. The broken line indicates the reflex threshold obtained by application of sound into the middle ear cavity. Note the improvement of the function up to 35 db.

Various pathological ear conditions were studied in the animal experiment as to their response to Middle Ear application of sound. The best functional results have been seen in Nerve type deafness. Slide No. 3 shows the diminished function of the cochlea due to an experimental Inner Ear lesion. When sound is applied through an opening of the Bulla Ossea, the cochlea shows improvement of function up to 30 and 35 db. Lesions which

resemble Otosclerosis can be produced experimentally in animals, by application of plaster of Paris in the oval window region. When the stapes-foot plate is immobilized the functional gain by Middle Ear application of sound is not as great as in Inner Ear deafness.

Our observations on patients have shown us that the phenomenon also can be demonstrated in the human. The human cochlea is able to receive sound energy conveyed through a mastoid fistula. The "hearing gain," however, is not as outspoken as in the animal. Audiometric tests revealed improved hearing of 5-10 db. This improvement was not considered great enough to carry out more fistula operations.

The fact that Doctor Dixon is able to show us here patients in whom he observed apparently far greater improvements, is very remarkable.

O. JASON DIXON, M.D., in closing: I am sorry that time did not permit me to show some statistical data, although I always dislike statistical data very much and get around it if I can. I feel that I am among friends. I am also from Missouri. I had to be shown and I invite you to Missouri to be shown. You are all welcome in my operating room. My patients are getting along well. I am not afraid to have them meet each other.

So far as running audiograms are concerned, I have run audiograms. I forgot to look at them. I still think the tuning fork is all right. After all, there is one thing that these patients want, that is to hear. They just want to hear one thing. That is the spoken voice. Never in the history of this work have people so desired to hear the spoken voice, because we have the talking movies and we have the radio. Probably the most grateful patient I have, I have done the least for, a little woman thirty-five years old, a teacher in a deaf school, who finished her last two years in college and took her master's degree when she was totally deaf. She learned lip reading and copied notes from her friends and made wonderful progress. She finished at Wisconsin. The only thing I have done for that lady is when she calls up her friend and says, "Mary, I will be over in thirty minutes," she can hear Mary say, "Don't come. I am leaving to go shopping." Before that she just said," I will be over," and she went whether or not Mary was there. Now she can talk over the telephone.

These patients present very much of a psychic problem. I cannot be seventeen doctors in one. I am not a physicist; I am not a physiologist. I like to think of how this works. By the way, there is a lady here in the house who told me more about the physiology of it and the physics of it than anybody to whom I have talked or anything I have read.

Otosclerosis as far as I am concerned is out of the window. Some of the best results I have had have been diagnosed as otosclerosis. I am getting repeaters of patients coming back whom I saw five, six or seven years ago and told to save their money. No longer should we say to a patient, "What cannot be cured must be endured." I think that is all over. I do not mean to say that I can cure all of them. I certainly cannot. Some of my greatest surprises have come in cases that I thought I stood little chance to cure and some for whom I hoped to do the most I have done the least. I tell each one of them that I do not know. I tell them that it will take an hour and a

halt to do the operation. I let them see other patients and tell them about the risks, that I will have to operate near the brain, near the facial nerves, and near the lateral sinus. I tell them I cannot do any harm and you never know until you try. I tell them to go out and talk to some of these other people. Most of our patients have been doing that. They have been getting a line on us before they ever come to our offices.

I am particularly grateful to my otological friends in Kansas City. Practically every one of them has sent me at least one good case. I am also grateful to the doctors in other cities who have tried me out and I apologize for some of the heart-breaking disappointments that I have had in those very fine cases. But in no case am I discouraged. Strange as it may seem, in about 35 per cent of the cases the patient is hearing just as well in the other ear, if not a little better. I would not have stated it, if Doctor Kopetzky had not already stated it. In the audience now is a patient who had that experience.

The case I like is the one that comes in wearing a hearing aid. Nobody wears a hearing aid if he can hear the spoken voice. I have no fight to pick with hearing aids. I like to see them because some of my patients who are rather aggressive will even grab them off the street and bring them up. The patient with the hearing aid has proven that he needs help. He is trying. He is dissatisfied. He comes in and he is grateful for a little help. In all of the cases where they have had the conductive type of deafness, where they have gotten prompt results (and that has been practically all of them), they have discarded the hearing aids. Three of my pateints sold hearing aids. This man sitting in the audience wore one for seven and one-half years. He came to Chicago wearing it.

I like to have the patient thoroughly investigate it and go home and think it over, because it is a tremendous responsibility on my part. It is also a tremendous responsibility to refuse. The patients of whom I am afraid are the hypertensive, the diabetic, the brain tumor, or the pathological vascular lesion in the ear. So far I have had no tragedies. I have had erysipelas and incidental infection in my patients, two of whom threatened to commit suicide because they lost their hearing temporarily. Just in the last year three patients who had given up as having no cure came in and apologized for all the things they thought.

I like to have them come in with an interpreter to take notes. The lip readers are not so good as a rule because, after all, they are still lip readers.

But this offers something in a very modest way, without risk, with only about four days in the hospital, and whether it works or not, certainly the patient will be made no worse. How to select them further than that I think depends upon our usual judgment by which we select patients. Of my two very best cases one is a man 67 and one is 64. Age is no factor. I was callous enough last month to turn down a woman who weighed 270 pounds. She is a telephone operator, who is sending her girl to high school, and is about to lose her position. I say it is very gratifying to see these smart people, who were losing their positions, come back and even cry on your shoulder, saying that they are now back at work and are all right. They do not want to hear anything more about it. They like to forget it. When I do not hear from them, I know they are all right.

Instructional Subjects

The American Academy
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* * *

Forty-Fourth Annual Meeting
*Chicago * 1939*

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(With Particular Attention to the Fundamentals and the Clinical Application)

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I. Mucous membrane
   A. General considerations
   B. Essential constituents
   C. Epithelium
      1. Types
      2. Deviations: glands
   D. Stroma
      1. Constituents
      2. Submucosa
   E. Supporting structures

II. Lymphatic tissues
   Waldeyer's ring

III. Nose and lacrimal apparatus
1. Olfactory mucosa
2. Septum
3. Turbinates
4. Sinuses
5. Lacrimal apparatus

IV. The pharynx
1. Nasopharynx
Eustachian tubes through middle ear
2. Oropharynx
3. Hypopharynx

V. Mouth
1. Lip
2. Cheek
3. Palate
4. Tongue
5. Salivary glands

VI. Larynx

VII. Trachea and bronchi

VIII. Esophagus

IX. Miscellaneous
Thyroid gland
Thymus gland

C-3. PHYSIOLOGY OF THE EAR AND ITS CLINICAL INTERPRETATION

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II. Fetal Stage

III. Congenital Defects

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Helenor C. Wilder, Washington, D. C.
T. L. Terry, M.D., Boston
Benjamin Rones, M.D., Washington, D. C.

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GEORGE R. CALLENDER, M.D., Col. U. S. Army (by invitation), and
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D-3. PATHOLOGICAL CHANGES IN THE CORNEA

BENJAMIN RONES, M.D., Washington, D. C.

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Faculty

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ROLLO MCCOTTER, M.D., Ann Arbor

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2. Anatomy of the Eyeball and Visual Pathway.
3. Anatomy of Eyelids, Muscles, and Lacrimal Apparatus.

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ROBERT VON DER HEYDT, M.D., Chicago

1. Corneal lesions
2. The anterior chamber

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Station Hospital, Mitchel Field, Long Island, N. Y.

Faculty

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ROBERT K. SIMPSON, M.D.
ELDRIDGE ADAMS, M.D.

OPHTHALMOLOGY IN AVIATION

FREDERIC H. THORNE, M.D., Lt. Col. M. C., U. S. Army

1. Outline of the history of ophthalmology in aviation.
2. Motility of the eye:
   a. Efficiency
   b. Anomalies
   c. Judgment of distances and factors influencing the efficiency of this faculty
   d. Bearing on the efficiency of the pilot
   e. Optical principles involved and official methods of determination and —
   f. Qualification requirements of the armed forces.

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   a. Factors influencing, and qualification requirements of pilots and observers
   b. In low illumination-retinal adaptation
2. Field of vision for form and color
3. Central color vision
   a. Tests employed and qualification requirements
4. Errors of refraction and accommodation
5. Ophthalmoscopy
6. The ear, nose and throat examination
   a. Qualification requirements

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Chief, Medical Section, Civil Aeronautics Authority

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Minimum eye and ear requirements for certification in each grade — method of handling variations from required standard

Prescribed technique and equipment necessary in making examination

Frequency of required examinations

Variation of commercial examination from military

Outline of administrative procedure followed

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LEGRAND H. HARDY, M.D., New York
JOHN H. DUNNINGTON, M.D., New York

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HETEROPHORIA AND HETEROTROPIA

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J. H. ALLEN, M.D., Iowa City, Iowa

K. OCULAR PHYSIOLOGY

FRANCIS HEED ADLER, M.D., Philadelphia

The modern conception of retinal activity during light and dark adaptation.

L. GLAUCOMA

Faculty

1. HARRY S. GRADLE, M.D., Chicago
   Classification and Etiology

2. Georgiana Dvorak-Theobald, M.D., Chicago
   Pathology
3. Thomas D. Allen, M.D., Chicago
   Acute Incompensated Glaucoma
4. Samuel J. Meyer, M.D., Chicago
   Compensated Glaucoma
5. Louis G. Hoffman, M.D., Chicago
   Secondary Glaucoma
6. Peter Kronfeld, M.D., Chicago
   Pre-Glaucoma—Provacative Tests—Evaluation

M. NEURAL OTOLARYNGOLOGY
Marvin F. Jones, M.D., New York

N. CANCER OF THE LARYNX

Faculty

Chevalier Jackson, M.D., and
Chevalier L. Jackson, M.D., Philadelphia
a. Laryngofissure, with cadaver demonstration
b. Laryngectomy
c. Irradiation

O. THE FUNCTIONAL EXAMINATION OF HEARING AUDIOMETRY — HEARING AIDS
Scott N. Reger, Ph.D., Iowa City, Iowa (by invitation)

P. THE TREATMENT OF BRAIN ABSCESS
Joseph E. J. King, M.D., New York (by invitation)

R. THE CONSTRUCTION OF LIFE MASKS AND PROSTHESES

Faculty

Noah Fox, M.D., Chicago
Samuel Peluse, M.D., Chicago (by invitation)
John Harned, M.D., Chicago

S. POST-GRADUATE STUDY OF ANATOMY IN OTO-LARYNGOLOGY
Louis J. Birsner, M.D., St. Louis

SINGLE LECTURE COURSES

Ophthalmology

THOMAS D. ALLEN, M.D., Chicago
Uveitis: Malignant — Chronic

WALTER S. ATKINSON, M.D., Watertown, N. Y.
Types of Anesthesia Practical in Ophthalmology

ARTHUR J. BEDELL, M.D., Albany, N. Y.
Fundus Photography

CONRAD BERENS, M.D., New York
Aniseikonia

A. B. BRUNER, M.D., Cleveland
Telescopic Lenses

FRANK E. BURCH, M.D., St. Paul
Methods for Cataract Surgery

WALTER E. CAMP, M.D., Minneapolis
Clinical Application of Pathology

FRANK D. CARROLL, M.D., New York
The Toxic Amblyopias

RAMÓN CASTROVIEJO, M.D., New York
Lesions of the Cornea — Surgical

C. P. CLARK, M.D., Indianapolis
Preparation of Cataract Patient

GRADY E. CLAY, M.D., Atlanta
Rare Retinal Lesions

FREDERICK C. CORDES, M.D., San Francisco
Anomalies — Congenital and Acquired — of the Optic Discs

SYBIL COX, Cleveland (by invitation)
Reading Difficulties

ARTHUR M. CULLER, M.D., Dayton
Vaccine and Fever Therapy

WINDSOR S. DAVIES, M.D., Detroit
Relation Between Clinical and Pathological Changes in Optic Nerve Head

WILLIAM THORNWALL DAVIS, M.D., Washington, D. C.
Presbyopia

EARLE B. FOWLER, M.D., Chicago
Endocrine Disturbances of the Eyes

A. D. FROST, M.D., Columbus, Ohio
Papilloedema

SANFORD R. GIFFORD, M.D., Chicago
The Treatment of Some Less Common Corneal Diseases

JOHN F. GIPNER, M.D., Rochester, N. Y.
Vascular Lesions and Changes in the Eye

TRYGVE GUNDERSEN, M.D., Boston
Vascular Lesions of the Cornea

PHILIP A. HALPER, M.D., Chicago
Finer Uses of the Cross Cylinder in Refraction

ERLING W. HANSEN, M.D., Minneapolis
Ocular Manifestations of Allergy

PARKER HEATH, M.D., Detroit
Ophthalmic Therapeutics

LOUIS G. HOFFMAN, M.D., Chicago
The Diagnosis and Treatment of Lid Conditions

EDWARD JACKSON, M.D., Denver
Experiments in Visual Optics

HAROLD H. JOY, M.D., Syracuse, N. Y.
Pupillary Changes of Diagnostic Value

P. J. LEINFELDER, M.D., Iowa City, Iowa
Neuro-Ophthalmology

ALBERT N. LEMOINE, M.D., Kansas City, Mo.
Treatments of External Diseases of the Eye

PHILIP MERIWETHER LEWIS, M.D., Memphis
Ocular Complications of Epidemic Meningitis

WALTER I. LILLIE, M.D., Philadelphia
Eye Changes Due to Intracranial Pathology

RALPH I. LLOYD, M.D., Brooklyn
Non-Suppurative Diseases of the Cornea

S. HANFORD McKEE, M.D., Montreal
Office Application of Ophthalmic Bacteriology

S. J. MEYER, M.D., Chicago
The Present Status of Retinal Detachment Surgery — Choice of Procedures

C. S. O'BRIEN, M.D., Iowa City, Iowa
Tumors of the Orbit — Differential Diagnosis and Treatment

LUTHER C. PETER, M.D., Philadelphia
Retinal Separation — End Results

MAX POSER, Rochester, N. Y. (Bausch & Lomb Company)
Technique in Scientific Microscopy and Photo-Micrography

ALGERNON REESE, M.D., New York
The Surgical Treatment of Primary Glaucoma

JAMES J. REGAN, M.D., Boston
Various Tests for Astigmatism

BENJAMIN RONES, M.D., Washington, D. C.
Corneal Dystrophies

C. W. RUTHERFORD, M.D., Indianapolis
Myopia — Etiology and Treatment

R. O. RYCHENER, M.D., Memphis
Affections of the Lacrimal Apparatus

JAMES S. SHIPMAN, M.D., Camden, N. J.
Hypermetropia — Methods of Handling

ALBERT C. SNELL, M.D., Rochester, N. Y.
Medicolegal Ophthalmology

EDMUND B. SPAETH, M.D., Philadelphia
Indications for the Technique of Various Surgical Procedures in the Correction of Ptosis

SCOTT STERLING, Rochester, N. Y. (by invitation)
Bifocals and the Selection of Bifocal Types

EDWARD STIEREN, M.D., Pittsburgh
Industrial Ophthalmology, Including Traumatic Industrial Surgery and Removal of Foreign Bodies

GEORGE H. STINE, M.D., Colorado Springs, Colo.
Diagnosis and Localization of Retinal Tears

ARNO E. TOWN, M.D., New York
Contact Lenses

DERRICK VAIL, M.D., Cincinnati
Opto-Chiasmatic Arachnoiditis

HENRY P. WAGENER, M.D., Rochester, Minn.
Differential Diagnosis and Types of Retinitis

CLIFFORD B. WALKER, M.D., Los Angeles
The Refinements of Perimetry

WEBB W. WEEKS, M.D., New York
Complications of Cataract Surgery

MEYER WIENER, M.D., St. Louis
Lid Lesions and Methods of Surgical Correction

* * *

Otolaryngology

JAMES A. BABBITT, M.D., Philadelphia
Endaural Surgery in Middle Ear and Mastoid Infection

JOHN F. BARNHILL, M.D., Miami Beach, Fla., and
WILLIAM MELLINGER, M.D., Santa Barbara, Calif.
Structures in the Fossae of the Skull Which Have Important Relations to Ocular, Aural and Nasal Diseases

C. D. BLASSINGAME, M.D., Memphis
Modern Ideas in Office Practice

C. C. BUNCH, M.D., St. Louis (by invitation)
The Functional Tests of Hearing

EDWARD H. CAMPBELL, M.D., Philadelphia
The Treatment of Chronic Progressive Deafness

LOUIS H. CLERF, M.D., Philadelphia
Diseases of the Esophagus

ANDERSON HILDING, M.D., Duluth, Minn.
Physiology of Upper Respiratory Tract

FREDERICK T. HILL, M.D., Waterville, Maine
Treatment of Chronic Suppurative Otitis Media

A. R. HOLLENDER, M.D., Miami Beach, Fla.
Physical Therapeutic Procedures in Otolaryngology

A. J. CONE, M.D., St. Louis
A Critical Study of Sinus Disease as an Etiological Factor in Serious Eye Disorders; A New Method of Evaluation by Laminagraphs

JAMES B. COSTEN, M.D., St. Louis
The Mandibular Joint Syndrome: Anatomy, Pathology, Diagnosis and Treatment

WALLACE DEAN, JR., M.D., St. Louis
Laboratory Aids in the Diagnosis of Otolaryngological Conditions

BERT E. ELLIS, M.D., Indianapolis
The Treatment of Intranasal Tumors

MATTHEW S. ERSNER, M.D., Philadelphia
The Value of Sulfanilamide and Sulfapyridine in Otorhinolaryngologic Conditions

I. FRIESNER, M.D., New York
Intracranial Complications of Ear Disease

FRENCH K. HANSEL, M.D., St. Louis
Allergy of the Nose and Paranasal Sinuses

LYMAN H. HEINE, M.D., Fremont, Nebr.
Examination of the Patient

LEE M. HURD, M.D., New York
Physical Therapy in Otolaryngology

JOSEF WEINTRAUB, M.D., Cincinnati (by invitation)
I — The Connective Tissues of the Neck (A New Interpretation)

SAMUEL IGLAUER, M.D., Cincinnati
II — Deep Infections of the Neck and Mediastinum

P. E. IRELAND, M.D., Toronto
Surgical Emergencies in Laryngology

CHEVALIER JACKSON, M.D., and
CHEVALIER L. JACKSON, M.D., Philadelphia
Direct Laryngoscopy: Indications and Technic with Special Reference to Biopsy and Removal of Benign Tumors

SAMUEL J. KOPETZKY, M.D., New York
Diagnosis and Therapy of Otogenic Systemic Infections

ROMEO A. LUONGO, M.D., Philadelphia
The Fronto-Ethmo-Sphenoidal Operation

E. M. MACEWEN, M.D. (by invitation) and
E. W. SCHELDRUP, M.D. (by invitation), Iowa City, Iowa
Applied Anatomy of the Neck

A. S. MACMILLAN, M. D., Boston (by invitation)
X-Ray Examination of the Sinuses and Mastoids

PAUL M. MOORE, JR., M.D., Cleveland
External Otitis and Abnormalities of the External Ear

HARRIS P. MOSHER, M.D., Boston
Esophagus

HORACE NEWHART, M.D., Minneapolis
Audiometers and Hearing Aids, Their Scope, Limitations and Practical Application

LYLE S. POWELL, M.D., Lawrence, Kans.
The Laryngo-Stroboscope in Clinical Examination of the Larynx

JOEL J. PRESSMAN, M.D., Los Angeles
Physiology of the Larynx

ARTHUR PROETZ, M.D., St. Louis
Practical Management of the Difficult Sinus Case

LYMAN RICHARDS, M.D., Boston
Diagnosis and Practical Treatment of Sinus Disease in Children

JAMES MILTON ROBB, M.D., Detroit
Tonsillectomy and Adenoidectomy and Their Complications

LYLE M. SELLERS, M.D., Dallas, Texas
Acute Otitis Media

BENJAMIN H. SHUSTER, M.D., Philadelphia
Vertigo

FERRIS SMITH, M.D., Grand Rapids
Surgery of the Fronto-Ethmo-Sphenoid Sinuses

JOSEPH A. SULLIVAN, M.D., Toronto
Facial Nerve Repair

WALTER H. THEOBALD, M.D., Chicago
Pitfalls in the Practice and Treatment of Otolaryngology

GABRIEL TUCKER, M.D., Philadelphia
I — Bronchology—Discussion of the Interpretation of Bronchoscopic Findings in Lesions of the Tracheobronchial Tree
II — Gastroscopy from the Viewpoint of the Broncho-Esophagologist

O. E. VAN ALYEA, M.D., Chicago
The Ethmoid Labyrinth

GEORGE L. WHELAN, M.D., Philadelphia
Surgical Anatomy of the Ethmoidal Labyrinth

WILLIAM F. WHELAN, M.D., Philadelphia
The Anatomy of the Temporal Bone

HENRY L. WILLIAMS, M.D., Rochester, Minn.
The Diagnosis and Treatment of Petrositis

Motion Pictures
and
Instruments

The American Academy
of Ophthalmology and Otolaryngology

Forty-Fourth Annual Meeting
Chicago • 1939

Motion Pictures and Instruments

SECRETARIES IN CHARGE

W. L. BENEDICT, M.D., *for Ophthalmology*
J. L. MYERS, M.D., *for Otolaryngology*

PRESIDING OFFICERS

C. A. BAHN, M.D., *for Ophthalmology*
W. J. MELLINGER, M.D., *for Otolaryngology*

OPHTHALMOLOGY

PROGRAM

Kodachrome Clinic of Rare and Interesting Anterior Eye and Fundus Pictures

ROBERT VON DER HEYDT, M.D., Chicago, Ill.

The following color photographs of actual cases were shown and discussed:

FUNDUS CONDITIONS

1. Large coloboma involving part of nervehead.
2. Cyst floating in the vitreous.
3. Choroiditis guttata ("Druesen der Glaslamelle").
4. Oguchi's disease — congenital nightblindness.
5. Cyanosis retinae.
6. Retinal detachment due to contraction of bands in the vitreous.
7. Vitreous detachment.
8. Anastomosis of veins.
9. Hemorrhages and obliterated vessels in hypertension.
10. Gumma at the nervehead.
11. Periphlebitis.
12. Kuhnt-Junius macular degeneration. (2 cases.)
13. Papilloretinitis with macular change.
14. Retinitis exudative externa (Coat's disease).
15. Retinochoroiditis juxtapapillaris (Jensen's disease).
16. Venous thrombosis.
17. Embolism (4th and 8th days).
18. Hole in the macula.
19. Healed choroidal lesion.

20. Healed tubercle in macula.
21. Healed tubercle with reactivation at margin.
22. Massive sarcoma of choroid. (2 cases.)

EXTERNAL DISEASES

1. Sulcus marginalis senilis (senile marginal groove).
2. Venous congestion of eyeball in cavernous sinus thrombosis.
3. Interstitial Keratitis (incipient "salmon patch").
4. Old interstitial, "palm leaf" lesion.
5. Scleral ulceration.
6. Essential atrophy of iris. (2)
7. Haemophthalmos — blood — pus and crystals in anterior chamber.
8. Epithelioma at the limbus.
9. Discform Keratitis. (3 stages.)
10. Cataracta complicata.
11. Annular superficial hyaline degeneration of cornea.
12. Pigmented corneal lesion in osteitis deformans.
13. Fuch's epithelial dystrophy due to cornea guttata.
14. Sclerestasia.
15. Blood tinging of cornea. (2 stages.)
16. Window transplant (Dr. Castroviejo) 2 years old.
17. Glaucoma capsulare.
18. Siderosis. (3)
19. Chalkosis.
20. Vitreous hernia with blood in suspension.
21. Pearl cyst — eyelash in anterior chamber.

The Refraction Department of the Wills Hospital

I. S. TASSMAN, M.D., Philadelphia, Pa.

Contact Lenses

G. VICTOR SIMPSON, M.D., Washington, D. C.

Contact Glasses

MR. HUGH HUNTER (Belgard-Spero, Inc.), Chicago, Ill.

Some Varieties of Ocular Nystagmus

J. E. LEBENSOHN, M.D., Chicago, Ill.
R. F. CARMODY, M.D., Gary, Ind., and
S. SUGAR, M.D., Chicago, Ill.

A. *Keratoplasty*

MAX FINE, M.D., and
HANS BARKAN, M.D., San Francisco, Calif.

B. *Shortening of the Eyeball*

W. E. BORLEY, M.D., and
HANS BARKAN, M.D., San Francisco, Calif.

A. (1) *Corneal Transplant*
(2) *Case of Pulsating Exophthalmos in Neurofibromatotis*

B. (1) *Free Transplantation of Tarsus for Reconstruction of Upper Lid Border*

(2) *Free Transplantation of Tarsus for Reconstruction of Lower Lid*

C. *Diathermy for Retinal Detachment (Lacarerre Technique)*
WENDELL L. HUGHES, M.D., Hempstead, L. I.

A. *Orbitotomy for Tumor of the Orbit*

B. *Trephine for Glaucoma Simplex*
W. L. BENEDICT, M.D., Rochester, Minn.

OTOLARYNGOLOGY

PROGRAM

The Operative Technic for the Surgery of Chronic Progressive Deafness
W. W. MORRISON, M.D., New York, N. Y.

New Operation for Conductive Deafness
O. J. DIXON, M.D., Kansas City, Mo.

A Photographic Study of the Larynx
JOEL J. PRESSMAN, M.D., Los Angeles, Calif.

Surgical Repair of Old Nasal Fractures with Dislocated Septum (in color)
JACQUES W. MALINIAC, M.D., New York, N. Y.

Phylogenetic Development of the Tonsil
JAMES M. ROBB, M.D., Detroit, Mich.

Radical Mastoid Operation with an Improved Technique and an Original Flap
LOUIS DAILY, M.D., Houston, Texas

A. *Laryngofissure, Jackson Anterior Commissure Technique*

B. *The Voice After Laryngeal Operations*
C. L. JACKSON, M.D., Philadelphia, Pa.

Malignant Tumors
GABRIEL TUCKER, M.D., Philadelphia, Pa.

Various Types of Benign Tumors of the Larynx
GORDON B. NEW, M.D., Rochester, Minn.

High Speed Pictures of the Vocal Cords
Courtesy of MR. JOHN MILLS, Bell Telephone Co. Laboratories, New York, N. Y.

A. *Lateral Transthyroid Pharyngotomy* (in color)
B. *Laryngectomy*
H. B. Orton, M.D., Newark, N. J.

The Treatment of Tubercular Ulcers of the Tracheo Bronchial Tree Through the Bronchoscope
Arthur Palmer, M.D., New York, N. Y.

Fundamentals of Technique for Acute Surgical Mastoiditis
Lawrence R. Boies, M.D., Minneapolis, Minn.

AN EYE SPECULUM FOR INFANTS*

CONRAD BERENS, M.D.
NEW YORK

THE SPECULUM to be described for use in operations on infants is similar to the speculum described in 1932[1] for adults. However, the coil spring, originally used, has been replaced by an inside spring, and the eyelid plates are smaller and no longer movable. The arms of the forceps have been made lighter and thinner than in the adult model.

The length of the eyelid plates is 12 mm. and the maximum expansion of the plates is 22 mm. (Fig. 1).

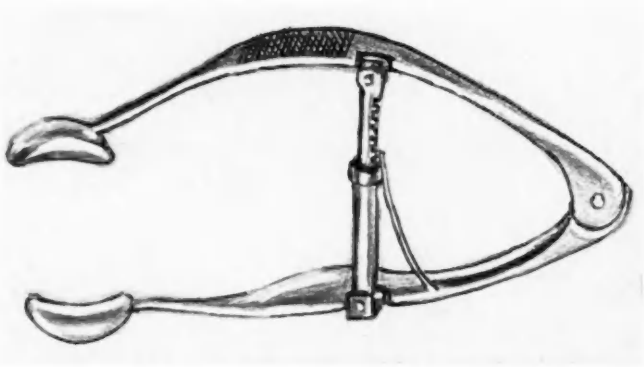


FIG. 1 — (Berens) — Eye speculum for infants.

The apparent advantages of this speculum are that (1) the eyelashes and the meibomian glands are excluded from the field of operation. (2) There are no protrusions upon which the sutures may become entangled. (3) The special catch used for locking the instrument in any desired position without the use of a screw permits the speculum to be inserted and removed with one hand.

* Aided by a grant from the Ophthalmological Foundation, Inc.
Made by V. Mueller, Inc., Chicago, Illinois.

(4) The arms of the speculum conform well to the features. (5) The size of the eyelid plates and the light weight makes the instrument especially adapted for use in infants.

REFERENCES

1. Berens, C.: *An Eye Speculum*, Trans. Sect. Ophth., American Medical Association, 1932, p. 357.

A CHART FOR TESTING THE VISUAL ACUITY OF AMBLYOPIC CHILDREN AND ADULTS AND A KINDERGARTEN VISUAL ACUITY CHART*

CONRAD BERENS, M.D.
NEW YORK

THE ORIGINAL drawings of the chart for testing visual acuity of amblyopic children and adults and the kindergarten visual acuity chart have been presented[1] but they have now been printed† (Figs. 1, 2 and 3). In the printed form these charts have been found to be practical in office and clinic use.

REFERENCES

1. Berens, C.: *A Chart for Testing the Visual Acuity of Amblyopic Children and Adults,* Brit. J. Ophth. 21:661, 1937; *Kindergarten Visual Acuity Chart,* Am. J. Ophth. 21:667, 1938.

* Aided by a grant from the Ophthalmological Foundation, Inc.
† Published by the American Optical Company.

FIG. 1 — (Berens) — Front view of the chart for testing the visual acuity of amblyopic children and adults.

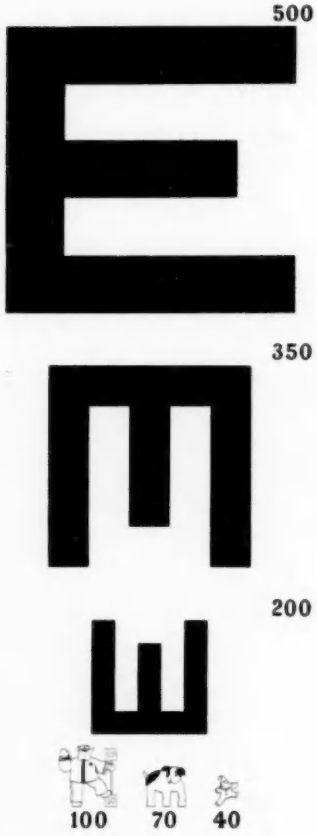


FIG. 2 — (Berens) — Back view of the chart for testing the visual acuity of amblyopic children and adults.

FIG. 3 — (Berens) — Kindergarten visual acuity chart.

ANOTHER FORCEPS FOR INTRACAPSULAR CATARACT EXTRACTION*

RAMON CASTROVIEJO, M.D.
NEW YORK

FOR SOME 12 years I have been employing forceps for intracapsular cataract extraction. Several types of forceps which I have used contained many good features, but none embodied all of the features which I considered necessary for ideal satisfactory performance.

Combining details taken from some of the forceps, and adding others which none of them had, I designed another instrument which after trial in over fifty operations for cataract, was found to perform in my hands better than those previously used.

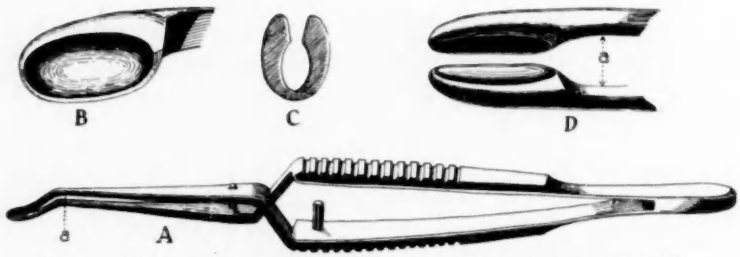


FIG. 1 — (Castroviejo) — A new forceps for intracapsular cataract extraction.

In presenting this instrument, I have purposely used the title "*Another* forceps for intracapsular cataract extraction," because I did not think that the few changes added to the combination of features already found in other forceps, justified the title of "*New* forceps for intracapsular cataract extraction."

The characteristics of the forceps here presented are as follows: The biting jaws are rounded, as illustrated in the figure, eliminating sharp edges or points which might prove injurious to

* From the Institute of Ophthalmology, Columbia Presbyterian Medical Center.

the intraocular structures, especially in uncontrollable movements of unruly patients. When closed, the biting jaws (Fig. 1-C) make contact only in that half (posterior) of the jaws which is used to grasp the capsule. The other (anterior) half of the jaws do not come together, in order to eliminate the danger of grasping the iris at the same time as the capsule. (Those operators who are accustomed to deliver the lens by the tumbling method may be hampered by the complication of an iris which is caught in the jaws of the forceps, especially in patients whose pupils do not dilate well.) The anterior half of the jaws is smoothed out and uniformly curved throughout, in order to diminish as much as possible the danger of injuring the corneal endothelium. When the jaws are closed the branches of the forceps are 1 mm. apart (A-a and D-a) to eliminate the possibility of grasping the iris with this portion of the forceps.

The curvature of the branches has been made to conform with that of the cornea. This makes easier the manipulation of the forceps within the anterior chamber, especially for those surgeons who are accustomed to grasp the capsule of the lens below the center of the pupillary area.

Cross-action has been incorporated in this model of forceps to reduce the tension of the operator's fingers upon the forceps, and thus to increase his sense of touch while the zonular fibers are being ruptured.

The cross-action also controls the opening of the jaws to a maximum of 6 mm., enough to obtain a good, but not excessive bite of the capsule, reducing the complication of a ruptured capsule, especially in swollen lenses.

The tendency to exert too much or too little pressure, thus crushing the capsule, or releasing it unconsciously at the most inoportune moment of the delivery, has been removed in the present cross-action forceps.

My experience with this model of instrument has convinced me that the lens capsule is grasped firmly enough to assist in the rupture of even the most resistant zonular fibers, and yet not too firmly to tear or crush the capsule.

I wish to acknowledge my indebtedness to Kalt, Elschnig, Arruga, Verhoef and Sinclair, from whose forceps I have taken some of the features incorporated in the instrument here described. I also wish to express my appreciation to C. R. Storz for his cooperation in making the instrument.

THE OPTOKINETOSCOPE

ALBERT C. SNELL, M.D.
ROCHESTER, NEW YORK

THIS is a simple instrument that any amateur can make. It consists of a vertical drum about 25 cm. in height and 19 cm. in diameter. On the drum are painted alternate black and white vertical stripes each 25 mm. wide. A central rod is fitted through the vertical axis of the drum so that it may be revolved slowly about this axis. A suitable handle is placed just underneath the drum into which the center rod may be fastened. The instrument is similar to a primitive kymographion.

By slowly turning the drum before the eyes of the subject, either from the right or from the left, nystagmus may be induced in seeing eyes. This is called optokinetic nystagmus and may be defined as the occurrence of nystagmus-like twitchings or movements of the eye when the subject looks at moving objects. This is the common phenomenon which occurs, for example, when looking at outside objects through the window of a train or moving vehicle. The optokinetic nystagmus is explained by the fact that during movements of either subject or object the individual can see objects only when the eye is fixed momentarily on one object after another.

To make the clinical test the instrument is held before the examinee's eye or eyes at the usual reading distance or somewhat further away, and the examinee is then asked to look at the stripes and try to see if he can tell whether they are moving or standing still. It is most difficult for a seeing person to avoid looking at the device and when he does so the nystagmus will be induced.

This is a comparatively simple objective test for unmasking simulated or hysterical blindness of one or of both eyes. When only one eye is alleged to be blind the good eye must be occluded while making the test. The presence of optokinetic nystagmus is proof that the tested eye is not blind.

A SIMPLIFIED OPERATION FOR KERATOPLASTY

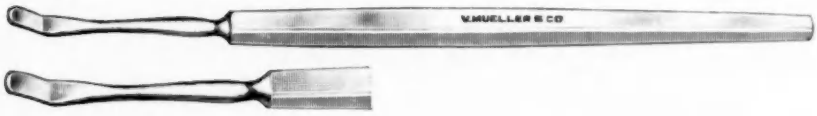
A. S. Green, M.D.
SAN FRANCISCO

Transplanting clear corneal tissue from an enucleated eye to an opaque eye has never developed any real popularity in the past, mainly because there were so few successful results. There were two principal reasons for the failure; the first, and most important, being the difficulty of obtaining healthy, clear corneas, the second the very complicated technic.

These difficulties have been largely solved by the epoch-making contributions of Professor Filatov of the U.S.S.R. He discovered that a section of cornea taken from a healthy patient who had just died as the result of a fatal accident could be used successfully for the transplanting in a favorable eye. He later found that by keeping the eye under aseptic conditions for two or three days at a temperature of about four degrees centigrade, the results were even better than when used immediately after enucleation. This solved the problem of obtaining sufficient desirable material whenever needed. He also simplified the method of keeping the graft in place without putting sutures into the corneal tissue itself, by using a flap of the full width of the upper conjunctiva and everting it so that the epithelial surface only, touched the corneal wound and retaining it by suturing it to the lower conjunctiva. Fuller details with new instruments and a simplified technique will be published shortly.

A CORNEO-CONJUNCTIVAL DISSECTOR

A. S. GREEN, M.D.
SAN FRANCISCO



To SIMPLIFY an operation should be the aim of all surgery. It is axiomatic that the more complicated the operation, the more trauma produced. Insult to the tissue interferes with healing and may even defeat the purpose of the operation. These remarks apply particularly to ophthalmic surgery. In trephining for glaucoma an especially delicate step is that of dissecting down the conjunctival flap and splitting the cornea so that about 1 mm. of the bluish corneal tissue shows at the end of the dissection. This is necessary to insure entering the anterior chamber. Many operations in trephining for glaucoma result in failure because this is not attained before applying the trephine.

To our knowledge, there has been no adequate dissector available up to the present time. We have used the tips of scissors, as used by Colonel Elliot. For many years we employed a small keratome with the point blunted and rounded off.

While in Australia a few months ago I was presented with a dissector which embodied a very important feature, viz., the end was beveled off like a chisel. We incorporated this idea in the instrument shown above. This instrument has a blade which has a slight concavity on the under surface to fit over the convexity of the eyeball. The dissecting end is beveled so that the tendency is to cut down toward the sclera and cornea and away from the conjunctival flap, thus helping to avoid perforation. As large a corneal crescent may be exposed as desired.

THE PURKINJE FIGURE AS APPLIED TO GIVE AN ABSOLUTE AND DIFFERENTIAL TEST OF THE PRESENCE OF SEPARATED RETINA AND THE NEON LIGHT PERSISTENT TRACERY IMAGE AS USED TO TEST RIGHT HANDEDNESS IN CONNECTION WITH RIGHT SEPARATION

CLIFFORD B. WALKER, M.D.
LOS ANGELES

LOGICALLY the separated retina cannot be expected to produce entopically the Purkinje vascular figure over the obviously elevated and detached portions. But the phenomena which I have noted and wish to emphasize as a reliable characteristic of separated retina in general is that the apparently undetached retina is also unable to produce the Purkinje vascular pattern as most conveniently tested by the recently developed common small tipped (5mm.) penstock handled flashlights.

USE OF THE PURKINJE FIGURE TO TEST FOR SEPARATED RETINA AND DIFFERENTIATE CERTAIN OTHER RETINAL FUNCTIONS

This simple way of producing the Purkinje figure by old fashioned flashlights was described by Eber and published in the *Journal of Ophthalmology*, Vol. 5, page 973, 1922, but has been little used. The pattern of the retinal vascular tree was elicited by rubbing the tip of a small flashlight against the outer ora serrata or equatorial zone of the eyeball through the closed lids, upper or lower, better lower. Other portions of the eyeball even the front can be used but with more difficulty and uncertainty. I do not agree that the picture is produced by means of rubbing only, as a simple stimulation to the retina, as described, because I find the use of vibrator machines normal to the eyeball (without motion parallel to the sclera) or any sort of motion perpendicular to the eyeball which might stimulate the retina plentifully will not bring forth the picture. But if there is enough movement at right angles

to vessels and parallel to the sclera so that there is enough lateral motion to slightly displace the shadow of the vessels with respect to the innermost retina, that is, with respect to the more fixed pigment layer holding percipient element tips, extending across the potential interval between the two primary layers where fluid accumulates in separated retina. Stimulation of the retina may not be necessary, only slight movement of vascular shadows must be produced to displace them with respect to the somewhat dark adapted retinal strip resulting from the usual pupillary direction of light. Then normally after a few ¼" strokes at ½ sec. frequency the complete picture flashes up instantly throughout in the manner of an all or none reaction. A dark room is not always required for soon the patient learns to see the figure in ordinary room light. The normal shadow picture of the vessels from light through the pupil has simply been suppressed previously in the interest of safety.

The patient soon learns to see this pattern (with one or even both eyes open) superimposed upon his total or usual vision. If superimposed upon screen or perimeter usually the character of the branching at various degrees of field may be approximated. As soon as we have taught the patient to see the Purkinje figure by method described and also at the same time the flash of the moving Neon light, we may combine the two *with the eye open* and see the Neon image (now a bluish-green due to retinal fatigue to red) superimposed upon the redish inverted Purkinje picture (as a screen) actually move upon the retina during the act of seeing (i.e., the patient gets the impression of seeing his retina "*see*"). This makes a good test to differentiate separated retina from any other condition where the field of vision may have scotomatous, sector or hemianoptic defects. The blinded separated retinal area and all the rest of the retina will still give the red background, but not the vascular pattern of Purkinje. Further, the elevated retina will see the green Neon image moving where large perimetric test objects could not be seen, unlike the blinded areas in glaucoma or large sector defects for instance. Yet the separated retina can never see the dichotomous branching or any part of the vascular picture even in the apparently undetached seeing portions and even in the first week of duration. Perhaps as soon as a hole occurs in the retina, fluid evidently saturates the space available between the two primary layers, putting nerve fibers on a stretch and sufficiently increasing the distance between vessel and percipient tips to blur the delicate shadow picture (Purkinje) beyond rec-

ognition although this undetached retina still functions rather normally while the nerve elements are not actually uprooted. There may be a still earlier stage where part of the figure is present.

Even the flatest type of separated retina fails to show any vessels of the Purkinje figure because the slightest film of edema in the potential space between the two primary retinal layers (crossed only by percipient element tips) will increase the penumbra of vascular shadows beyond recognition. It would be easy to carry these tests too far as regards moving the eye back and forth, etc., to see the Neon flash. Therefore, this is reversed and the test light is moved. The patient is required to hold the eyes open and fix on something straight ahead without winking much or the lids are held open for him if they interfere. But to control the pressure of my other Purkinje test, I have devised this little light device to be used with any spring tenometer. I prefer the Sobansky, a very accurate, durable instrument from the Hans Lauber clinic. The light (bronchoscopic) rides under the foot plates and it may be connected to the cut off, the light or ring a bell or buzzer at the tension reading for that individual. Very little pressure is used. Usually just enough to slide the lid on the sclera is necessary. The frequency of the motion is about 2 per second for a continuous picture, with such amplitude as the skin looseness permits (⅜″).

In case of a separation of the choroid the Purkinje figure still persists but I have found it absent in advanced retinitis pigmentosa. Sometimes a hematoma or hemorrhage may imitate a tumor ophthalmoscopically and transillumination with a Lange lamp is black as a tumor. Here it is well to teach when possible the simultaneous observation of the Purkinje picture while transilluminating with the Lange lamp. From one case only I judge the Neon lamp will appear as a green blur over hemorrhage in the choroid. I have not yet found a tumor early enough to test differently than a separated retina. In all other lesions of retina and optic pathway I have tested recently, the differentiation has been sharp, showing neither figure or Neon light in perimetric field defects.

THE PERSISTENT IMAGE TEST FOR HANDEDNESS WITH VAPOR LAMP (NEON)

If a small ¼ to ½ watt Neon vapor pilot lamp 10 to 20 feet distant is observed in a darkened room it will be noted that in the horizontal meridian the persistent image — an interrupted streak

of (red) light like a dashed line,* is seen to move in the opposite direction to the rotation of the eye, thus reporting that the eye moves quite accurately in the horizontal line. But in the vertical meridian this does not hold. The persistent image usually dashes upward and to the left in a right handed individual who tries to sweep his eyes along a vertical meridian from the top(ceiling) of the fixation field to the bottom (floor) through the zone or location of the Neon lamp. The persistent image streak will make an angle to the left of the vertical as much as 40° up and to the left for a strongly right handed individual. The return trip will be more vertical.

In case the individual was born left handed and trained right handed first at home as regards holding the spoon and then at school as regards holding the pencil or pen, a phenomena analogous to stuttering occurs in this test. That is the persistent dashing line or streak may deviate in determining either right or left and a condition of ambidexterity exists. For a left handed individual the light deviates to the right. Yet I must say by way of exception that I have once had a purely left handed individual report a deviation to the right and also one instance where a right handed individual reported a deviation to the left.

In the great majority of individuals it seems clear that the eye has been habituated to helping the hand of choice especially in picking up or locating things to the right or left (lower quadrant, according to the handedness) in an automatic almost reflex manner. If the right hand for instance is picking up anything the eyes must turn to aid and even to anticipate the intended motion of the hand. This is cited only as an indication of the almost reflex manner in which the eye must aid the hand especially in the quadrant where the touch system so to speak is inefficient. This tendency of the eye to cooperate with the hand is evidently perfectly natural and probably originally even one of the factors in the survival of the fittest complex. The eyes turn down and out instead of straight down because that has been the general requirement for so long in the past to aid or cooperate with the handedness.

* The dark intervals are produced when the voltage of the ordinary 60 cycle 110 volt alternating current falls below 50 volts. This occurs 120 times a second. These lamps come in several colors from ¼ to 2 watts as pilot lamps for standard or automobile base. Several colors are obtainable but red is most satisfactory. The dashed persistent line is much easier to follow with peripheral vision than a solid line as from a flashlight because such streaks are being largely suppressed from ordinary light every night.

In this study it is clear that it seems to be one more indication that the hand and eye cooperate almost to the degree of a conditioned reflex and automatically assumed a dangerous position for weak retinas. That is to say consequently that after the eye has been turned into the down and out position an additional stimulation to the superior oblique muscle such as a jolt to the body, spinal column or head may sometimes result in just the exact tendonous jerk on the sclera in the upper temporal quadrant to rupture a cystic degeneration of the retina near the insertion of the superior oblique tendon. This first rupture perhaps being only a small hole usually in the blind retina may give the patient in the confusion of the moment, no visual symptom strong enough to lead him to recite the episode except after careful questioning. Yet on the other hand patients are apt to elaborate about circumstances not unimportant but connected with the discovery much later of a visual defect often not until the time when the macula is affected either by detachment or overhanging retina.

CONCLUSIONS

1. It is important to determine whether a patient is right or left handed or ambidextrous in clarifying the history with respect to etiological traumatism in Separated Retina especially in Group 1.
2. The weight of evidence seems to indicate that a jerk from the superior oblique tendon can, when the eye is turned down and out, produce a rupture near its insertion of a retinal cyst or weak retina forming the starting point for typical more or less triangular tears with legs extending along lines of low resistance toward the ora serrata.
3. This common type of traumatism is historically so difficult to elicit and prove that the resulting separation are often considered idiopathic, non-compensable accidents. Yet, as a matter of fact, they have often occurred in line of duty and would not have occurred if the occupation had been such that straight ahead or much less lateral turning of the eyes had been encouraged.
4. This viewpoint is perhaps subtle involving hydrodynamics and physio-psychology as well as anatomy and pathology or it would not require so much analysis. Yet without doubt much legitimate compensation has been lost to deserving individuals because sometimes the mechanism of an injury which breaks

a retina is so much more obscure than for instance an injury which breaks an extremity.

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Necrology

The American Academy
of Ophthalmology and Otolaryngology

Forty-Fourth Annual Meeting
Chicago • 1939

IN MEMORIAM

Benjamin Franklin Baer, Jr., Philadelphia, Pennsylvania
Charles B. Barker, Guthrie, Oklahoma
Bennett Sheldon Beach, New York, New York
Edward Jay Bernstein, Detroit, Michigan
Philip Frank Bernstien, Brooklyn, New York
Charles L. Billard, Washington, D. C.
Harry Philip Cahill, Boston, Massachusetts
Allan Rupert Cunningham, Halifax, N. S., Canada
Frank Embery, Philadelphia, Pennsylvania
E. Ross Faulkner, New York, New York
Lyman Robert Forgrave, St. Joseph, Missouri
Max Halle, New York, New York
Leslie Ramsey Hazlett, Butler, Pennsylvania
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John Mark Walsh, Rapid City, South Dakota
James Alfred Watson, Minneapolis, Minnesota

Benjamin Franklin Baer, Jr., M.D.

Doctor Baer was born in Philadelphia on November 4, 1879, and died there on December 19, 1938.

He received his early education at St. Luke's Academy and Central High School and shortly after his graduation from the University of Pennsylvania Department of Medicine in 1903, he became associated with Doctor George E. de Schweinitz, in the practice of ophthalmology, and this association lasted until the death of the latter.

Doctor Baer was Associate Professor of Ophthalmology at the University of Pennsylvania Medical School from 1904 until 1924, and later Attending Surgeon at the Wills Eye Hospital. From 1924 until the time of his death he was Associate Professor of Ophthalmology in the Graduate School of the University of Pennsylvania.

During the World War, Doctor Baer was a Captain in the Medical Corps, going overseas as Ophthalmologist to Base Hospital No. 20, at Chatel-Guyon, France, an organization which was recruited from the University of Pennsylvania. He skillfully conducted the extensive Eye service at this hospital, and in addition was frequently sent to the front on operating teams. On one of these trips he contracted "flu" and was invalided home in February, 1919, with the rank of Major.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American Ophthalmological Society and Philadelphia College of Physicians.

Doctor Baer was a most dynamic person with a large and diversified practice, quick in diagnosis and skillful in surgery.

He is survived by his widow, three daughters and two sons.

Charles Bolivar Barker, M.D.

Doctor Barker was born in Niobe, New York, on February 27, 1884, and died in Oklahoma City, Oklahoma, on February 12, 1939.

After receiving his early education from the public schools and Ewing College, he obtained his medical degree from Loyola School of Medicine in Chicago in 1912, and did post-graduate work in ophthalmology and otolaryngology in some of the large United States clinics. He began the practice of medicine in New Albany, Indiana, later moving to Guthrie, Oklahoma, to specialize.

He kept abreast with the newer practice and studied in Vienna in 1927, and in Cairo, Egypt, in 1932.

At the time of his death Doctor Barker was head of the eye, ear, nose and throat department and of the teaching staff of the Methodist Episcopal Hospital in Oklahoma City.

His hobby was farming and raising registered Hereford cattle. He was a 32nd Degree Mason.

Doctor Barker is survived by his wife and associate, Doctor Pauline Barker, three brothers and three sisters.

Bennett Sheldon Beach, M.D.

Doctor Beach was born on January 29, 1862, in Thomason, Connecticut, and died September 13, 1939, in New York City, having been a practicing physician in New York for over fifty years.

He received his M.D. from the College of Physicians and Surgeons, Medical Department of Columbia College, New York, in 1887, and began to practice medicine in New York. Later he specialized in ophthalmology and was for many years a surgeon on the staff of the New York Eye and Ear Infirmary until his retirement three years ago. He had also been at various times on the staffs of the Roosevelt Hospital, St. Mark's Hospital, physician in charge of Trinity Dispensary, and assistant ophthalmological surgeon to St. Bartholomew's Clinic.

Doctor Beach was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

He was a member of the Society of Colonial Wars and of the Sons of the Revolution and at one time was a major in the old 22nd Regiment, New York National Guard. He was a Mason.

He is survived by his widow and one brother.

Edward Jay Bernstein, M.D.

Doctor Bernstein was born in Baltimore, Maryland, on August 26, 1863, and died in Detroit, Michigan, on March 30, 1939.

He graduated from the University of Maryland as a Ph.G. in 1885, and from the College of Physicians and Surgeons of the University of Maryland with the degree of M.D., in 1887. Follow-

ing this he did post-graduate work in Johns Hopkins University School of Hygiene and Public Health, and later in Vienna, Berlin, Paris and London in post-graduate work in Otolaryngology.

He began the practice of medicine in Baltimore, later moved to Kalamazoo, and then to Detroit in 1917. He was at one time professor of otolaryngology at Woman's Medical College, Baltimore, and at various times attending ophthalmologist and otolaryngologist to the Bay View Hospital and the Freedman's Hospital in Baltimore, and Borgess Hospital, Kalamazoo, Michigan. He was formerly otolaryngological consultant to Grace Hospital in Detroit.

During the World War, Doctor Bernstein enrolled in the Volunteer Medical Service Corps.

He was a member of the American Academy of Ophthalmology and Otolaryngology, member and past president of the Maimonides Medical Society.

Doctor Bernstein was an ardent philatelist, and was Honorary Life Member and Editor of the Stampless Cover Division of the Philatelic Society.

He is survived by two sons.

Philip Frank Bernstien, M.D.

Doctor Bernstien was born in New York City on March 19, 1892, and died in Brooklyn on March 9, 1939, of heart disease.

He was a graduate of Syracuse University, with the degrees of B.S. and M.D., in 1917, following which he did post-graduate work in Berlin and Vienna in 1921 and 1922.

He began the practice of his specialties in Brooklyn and at the time of his death was Associate Otolaryngologist at the Brooklyn Jewish Hospital and head of the Outpatient Department. He was a member of the American Academy of Ophthalmology and Otolaryngology.

Doctor Bernstien served during the World War.

He is survived by his wife and three children.

Charles Louis Billard, M.D.

Doctor Billard was born in Washington, D. C., on May 13, 1878, and died there on May 1, 1939.

He obtained his medical degree from the Medical School of the University of Pennsylvania in 1904, and did post-graduate

work in ophthalmology at George Washington University. He later became Clinical Instructor in Ophthalmology at Georgetown University School of Medicine and George Washington University School of Medicine, and was at one time a member of the staffs of Providence Hospital, Gallinger Hospital, Children's Hospital, St. Elizabeth's Hospital, and the Episcopal Eye, Ear & Throat Hospital.

Doctor Billard was a member of the American Academy of Ophthalmology and Otolaryngology, local medical societies, and of Sigma XI, honorary fraternity.

His wife and one son survive him.

Harry Philip Cahill, M.D.

Doctor Cahill was born in Worcester, Mass., on October 4, 1884, and died in Boston on January 15, 1939.

He received his early education at Worcester Classical School and Holy Cross College, from which he graduated with the degree of A.B., magna cum laude, in 1907. After receiving his M.D. from Harvard Medical College, cum laude, in 1911, he spent several years in graduate research work in otology at the University of Basle in Switzerland, as a fellow from Harvard University. At the outbreak of the World War he returned to this country, served his internship at Boston City Hospital, and when the United States entered the war, went overseas where he served ten months as Captain.

At the time of his death Doctor Cahill was Surgeon-in-Chief at Boston City Hospital, Senior Surgeon at Massachusetts Eye & Ear Infirmary, Consultant at the Beth Israel Hospital and Lawrence Memorial Hospital. He was Professor of Otology at Tufts College and Assistant Professor of Otology at Harvard Medical School.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American Otological Society, and member of state and local medical societies.

In his youth, Doctor Cahill was a great athlete, a track and football star for Holy Cross, and the Cahill Trophy for the fastest one-mile college relay team is given in his memory. He was a keen research worker, a writer of a great many articles in his specialty of otology, a fine physician.

He is survived by his wife, his mother, and two sons.

Allan Rupert Cunningham, M.D.

Doctor Cunningham was born in Dartmouth, Nova Scotia, on May 8, 1881, and died at Saranac Lake, New York, on July 3, 1938.

He was educated at Halifax County Academy, Dartmouth, and received his medical degree at Dalhousie University, after which he did post-graduate work at Morsefield Hospital, London, England, and in Vienna. He returned to Halifax in 1906 and began the practice of his specialties.

At the time of his death he was assistant professor of diseases of the eye at Dalhousie University Faculty of Medicine, and on the staff of Victoria General Hospital, Halifax.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

During the World War, Doctor Cunningham served as Captain in the Royal Canadian Army Medical Corps. He was injured in the Halifax Explosion of December 6, 1917.

His wife and one daughter survive him.

Frank Embery, M.D.

Doctor Embery was born in Philadelphia on January 8, 1867, and died at his summer home at Southampton, Pa., on March 17, 1939.

He was a graduate of the Medico-Chirurgical College of Philadelphia with the degree of M.D. (summa cum laude), and did post-graduate work in Denver, Boston, and the Mayo Clinic, later practicing in Philadelphia.

Dr. Embery was a member of the American Academy of Ophthalmology and Otolaryngology, local and state medical societies, and was on the staff of the Frankford Hospital.

Dr. Embery was one of those straightforward, consistent otolaryngologists who commanded respect from his associates and patients. His routine and hospital service was of the highest order and he will be long remembered by his associates.

He is survived by his wife, two daughters and three grandchildren.

E. Ross Faulkner, M.D.

Doctor Faulkner was born in Glenholme, Nova Scotia, and died in New York City on May 29, 1939, at the age of sixty-three.

He received his A.B. and M.D. degrees from Dalhousie University, Nova Scotia, later going to London, England, where, in 1908, he was made a Fellow of the Royal College of Surgeons. He came to New York and began the practice of medicine in 1908, for a time taught anatomy and surgical pathology at the New York University Medical School, and later taught the same subjects at the Cornell Medical School.

When he decided to specialize on the ear, nose and throat, he became associated with the Manhattan Eye, Ear and Throat Hospital, where for the past sixteen years he has been a Surgical Director. He was also Professor of Otolaryngology at the New York Polyclinic Medical School.

He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, Active Fellow, formerly Vice-President and Member of the Council, of the American Laryngological Association, and a member of local and state medical societies.

To those who knew him well, Ross Faulkner was a rare and genial soul. Always modest by nature and increasingly reticent from his difficulty in hearing, he rarely responded to requests for public presentation from his wealth of experience, particularly in relation to the nasal sinuses. We have lost a man of still greater future promise.

He is survived by his wife and one sister.

Lyman Robert Forgrave, M.D.

Doctor Forgrave was born in Leon, Iowa, on September 26th, 1876, and died in Kansas City, Missouri, on March 9th, 1939.

He received his M.D. from Central Medical College in 1900, and took post-graduate courses in ophthalmology and otolaryngology in New York City and later in Vienna. At the time of his death he was on the staffs of the Noyes Memorial and Missouri Methodist Hospitals, and consultant to the State Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and on the Council from 1935 to 1938. He was an active and valued member of the Kansas City O. O. R. and L. Society, and of other local and state medical societies.

Doctor Forgrave was always genial and courteous, particularly gracious and helpful to the newer members correlating them to the new Academy activities. He was constantly at work advancing the interests of his chosen profession and perfecting himself in it.

His wife and two brothers survive him.

Max Halle, M.D.

Doctor Halle was killed in an automobile accident near Newcastle, Delaware, on September 7th, 1939, while returning from a visit to his son on a farm near Richmond, Virginia. He was 55 years old.

He was a graduate of Heidelberg and the Berlin University Medical School, and following his graduation became a professor at his alma mater. He came to this country four years ago, and has been rhinologist at the Polyclinic Hospital, member of the staff of the Medical Arts Center Hospital, and consulting otorhinolaryngologist at Broad Street Hospital, all of New York City. He was also on the courtesy staff of St. Luke's and Children's Hospital, Philadelphia, and has several times given postgraduate courses at Dr. Mackenzie's clinic. His office was at 667 Madison Avenue, New York.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

Doctor Halle was very well known for his work in plastic surgery, in which he became engaged after the war. He has published several books on the subject, and at the time of his death was engaged in writing another. He was particularly generous in demonstrating his particular surgical ideas in rhino-laryngology.

He is survived by his widow and one son.

Leslie Ramsey Hazlett, M.D.

Doctor Hazlett was born in Butler, Pennsylvania, on September 1, 1868, and died there on August 10, 1938.

He was a graduate of Jefferson Medical College in 1896, and began the practice of medicine in New Galilee, Butler County, Pennsylvania. Later he took post-graduate courses at the Polyclinic Hospital, Philadelphia, and the New York Post-graduate

Hospital, and took up the practice of his specialties in Butler, Pennsylvania, in 1914.

Doctor Hazlett was a member of the American Academy of Ophthalmology and Otolaryngology, and of local and state medical societies. At the time of his death he was on the staff of Butler Memorial Hospital.

He was greatly interested in all civic affairs and was a high degree Mason.

His widow, one son and one daughter survive him.

Edward Daniel Helfrich, M.D.

Doctor Helfrich was born in Galion, Ohio, on December 6, 1875, and died in Columbus, Ohio, on March 21, 1939.

He was a graduate of the Philadelphia College of Pharmacy and later took up the study of medicine, obtaining his medical degree from Ohio Medical University, Columbus, in 1902. He practiced medicine for some years in Galion, then took post-graduate work in New York City, Berlin and Vienna, and began the practice of eye, ear, nose and throat work in Columbus.

Doctor Helfrich was Professor of Applied Optics at Ohio Medical University, and instructor to the classes of nurses at Columbus Radium Hospital and Mercury Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

Doctor Helfrich was Captain in the Medical Corps during the World War, and served two terms in the Ohio Legislature from Crawford County. He was very much interested in civic affairs and was also Treasurer of the American Life Insurance Company.

His wife and daughter and two sisters survive him.

Benjamin Harrison Holmes, M.D.

Doctor Holmes was born in Burlington, Wisconsin, on March 10, 1938, and died in Racine, Wisconsin, on June 28, 1939.

He received his medical degree from Marquette University School of Medicine, Milwaukee, in 1913, and after his internship began the practice of medicine in Delevan, Wisconsin, later moving to Racine to specialize.

At the time of his death, Doctor Holmes was Chief of the Ear, Nose and Throat Department of St. Mary's Hospital and St. Luke's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

Doctor Holmes served during the World War as Captain in the Army Medical Corps.

He is survived by his wife and one daughter.

Walter Louis Horn, M.D.

Doctor Horn was born in New York City on March 1, 1891, and died there on December 29, 1938.

He received his early education in the public schools of New York and his medical degree from Fordham University in 1916. Following this he was an interne at Lenox Hill Hospital for two years, and resident physician at the Manhattan Eye, Ear & Throat Hospital for two years. He began the practice of otolaryngology in 1920.

At the time of his death Doctor Horn was Assistant Surgeon at the Manhattan Eye, Ear & Throat Hospital, Associate Otologist at Mt. Sinai Hospital, Surgical Director of Otolaryngology at Misericordia Hospital, and Assistant Oto-rhino-laryngologist at the Metropolitan Hospital.

For the past ten years Doctor Horn had been an Honorary Police Surgeon and was given a police Captain's funeral. He did a great deal of work for the Police Department and was credited with having aided them in official work.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and local and state medical societies.

Doctor Horn was a member of the Masons, Royal Arch Masons, and a golf enthusiast.

His wife and one son survive him.

Anders G. Hovde, M.D.

Doctor Hovde was born in Norway on September 30, 1875, and died in Los Angeles, California, on August 14, 1938.

He received his early education in the schools in Norway, then came to this country and obtained his B.S. degree from the Uni-

versity of Southern Minnesota in 1899, and his M.D. from the University of Minnesota School of Medicine, Minneapolis, in 1903. He practiced in Superior, Wisconsin, for many years, was on the staffs of the Biwabik and St. Mary's Hospitals, and lecturer to the Nurses' Training School. Later he took post-graduate courses in otolaryngology in Vienna, and moved to Los Angeles to practice his specialty.

At the time of his death Doctor Hovde was on the staffs of the Hollywood Hospital and the Queen of Angels Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and in local and state medical societies in both Wisconsin and California.

Dr. Hovde held a commission as Major in the Medical Section of the O.R.C.

He is survived by two sisters, and it is believed his mother is still living in Norway.

John Raymond Hume, M.D.

Doctor Hume was born in Milroy, Indiana, on May 3, 1883, and died in New Orleans, Louisiana, on October 29, 1938.

He received his medical degree from Indiana University School of Medicine in 1907 and served his internship in the State College Hospital in 1907 and 1908. He later took post-graduate work in otolaryngology, and began the practice of his specialties in New Orleans in 1914.

At the time of his death Doctor Hume was Professor of Otolaryngology and Head of the Department of Otolaryngology at the Tulane University of Louisiana School of Medicine, and had been for many years on the staff of the Eye, Ear, Nose and Throat Hospital and Presbyterian Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology and the American Laryngological, Rhinological & Otological Society; member and ex-president of the New Orleans Ophthalmological & Otolaryngological Society; member and ex-president of the Louisiana-Mississippi Ophthalmological & Otolaryngological Society; honorary member of the Texas Ophthalmological & Otolaryngological Society; member and secretary of the Ophthalmological & Otolaryngological section of the Southern Medical Association, and member of various other state and local medical societies.

Myron Ellis Kahn, M.D.

Doctor Kahn was born at Iron Mountain, Michigan, on January 8, 1892, and died in Chicago, Illinois, on May 25, 1939.

He received his M.D. from the University of Illinois College of Medicine in 1915, and began to practice medicine in Chicago. Later he specialized in ear, nose and throat work, and at the time of his death he was on the staffs of Children's Memorial Hospital, Michael Reese Hospital, and Winfield Tuberculosis Hospital, and Associate Clinical Instructor at the University of Chicago.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and of local and state medical societies.

During the World War, Doctor Kahn was a Lieutenant in the United States Navy Reserve, stationed at Great Lakes Naval Training Station.

He is survived by his widow.

George Charles Kreutz, M.D.

Doctor Kreutz was born in La Crosse, Wisconsin, on August 23, 1896, and died in Henry Ford Hospital, Detroit, on February 1, 1939.

He was a graduate of the University of Wisconsin and obtained his medical degree from Washington University School of Medicine, St. Louis, in 1924. Following his internship at the Missouri Pacific Hospital, St. Louis, and post-graduate work in otolaryngology with Doctor Barnhill in Indianapolis and Doctor Mosher in Boston, he went to Detroit to continue his training at the Henry Ford Hospital, where he entered the then combined division of Ophthalmology and Otolaryngology. Since 1936 he had been Surgeon-in-Charge of the Division of Otolaryngology of the Henry Ford Hospital, and was well known for his work in bronchoscopy and oesophagoscopy.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and local and state medical societies.

He had a diversity of interests outside of medicine, including music and sociology.

Dr. Kreutz is survived by his wife, one son and twin daughters.

William Humphrys Miller, M.D.

Doctor Miller was born on July 16, 1882, at Savannah, Ill., and died October 4, 1938, at Terre Haute, Indiana.

Following his early education at Morgan Park Academy in Chicago, he obtained his M.D. degree from Northwestern University Medical School in 1907, and did post-graduate work at Polyclinic Hospital in New York and at Barnes Hospital at St. Louis. He began the practice of medicine in Galena, Illinois, later moving to Terre Haute, Indiana, where he specialized in eye, ear, nose and throat work.

Doctor Miller was a member of the American Academy of Ophthalmology and Otolaryngology and of local and state medical societies. He was a member of the staff, and at one time President of the staff, of Union Hospital.

He served during the World War.

His wife, one son and one daughter survive him.

Frank Stiles Owen, M.D.

Doctor Owen was born in Medina, Ohio, on November 13, 1856, and died in Santa Barbara, California, on March 23, 1939.

After receiving his preliminary education at Oberlin College, he graduated from the University of Michigan Department of Medicine and Surgery in Ann Arbor in 1885. He began the practice of medicine in Stromsburg, Nebraska, in 1885, later took post-graduate work in otolaryngology in London and Paris, and returned to specialize in Omaha.

Doctor Owen was Professor Emeritus of laryngology and rhinology at the University of Nebraska College of Medicine, formerly oculist and aurist to the Immanuel Deaconess Institute and St. Catherine's Hospital, and rhinologist and laryngologist to the University Hospital, Omaha.

He was a life member of the American Academy of Ophthalmology and Otolaryngology, past presdient of the Omaha-Douglas County Medical Society, and member of other state and local societies.

His widow and four children survive him.

Harry Elmer Peterman, M.D.

Doctor Peterman was born in Indiana, Pennsylvania, on July 16, 1871, and died in Baltimore, Maryland, on February 25, 1939.

He received his early education at Teachers' Training College, Indiana, and his medical degree from Baltimore Medical College in 1895. He took post-graduate work in Berlin and Vienna, and began the practice of medicine in Baltimore, and was Associate Professor of Ophthalmology and Otology at Baltimore Medical College at the time of its merger with the University of Maryland.

Doctor Peterman founded the South Baltimore General Hospital and at the time of his death was its ophthalmic and aural surgeon, also visiting ophthalmic and aural surgeon to the Maryland General Hospital, and consulting ophthalmologist and otologist to the Frederick City Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons and member of local and state medical societies.

He served as member of Medical Advisory Board No. 2 of Baltimore during the World War.

Doctor Peterman is survived by his wife.

Robert Alexander Peterson, M.D.

Doctor Peterson was born July 8, 1892, at Palmer, Iowa, and died at Riverside, California, on January 27, 1939, as a result of nervous exhaustion and overwork due to the seemingly impossible conditions in China, while returning to his field of service as a medical missionary in Chengtu.

He received his M.D. from the Iowa College of Medicine in Iowa City, Iowa, in 1918, and became a medical missionary under the Board of Missions of the Methodist Episcopal Church, stationed at Chengtu, Szechwan, China, where he was professor of otolaryngology and associate in ophthalmology at the West China Union University, director of the Chengtu Eye, Ear, Nose and Throat Hospital, and consulting otolaryngologist to the Hospital for Men, and Hospital for Women and Children.

Doctor Peterson was a member of the American Academy of Ophthalmology and Otolaryngology, associate Fellow of the

American Medical Association, and Fellow of the American College of Surgeons.

He is survived by his widow, still living in Chengtu, West China, one brother and one sister in this country.

John Alsop Pilcher, Jr., M.D.

Doctor Pilcher was born in Petersburg, Virginia, on Dceember 21, 1904, and died in Roanoke, Virginia, on March 7, 1939.

His early education was gained at Virginia Polytechnic Institute, Blacksburg, Virginia, and his medical degree from the University of Virginia, following which he interned at Gill Memorial Eye, Ear, Nose and Throat Hospital, Roanoke, and Church Home and Infirmary, Baltimore. He took post-graduate work in otolaryngology at Bellevue Hospital, New York, and in bronchoscopy with Doctor Chevalier Jackson in Philadelphia.

He was a member of the American Academy of Ophthalmology and Otolaryngology, nad local and state medical societies, and at the time of his death was on the staff of the Gill Memorial Eye, Ear, Nose and Throat Hospital.

Doctor Pilcher was a Mason, member of the Roanoke Country Club and keenly interested in hunting, fishing and golf.

He is survived by his wife, his parents and two brothers.

Harry Sebastian Reger, M.D.

Doctor Reger was born in Indianapolis, Indiana, on October 28, 1877, and died in Jamestown, N. Y., on July 14, 1938.

He early attended the public schools of Ironton, Ohio, then the Collegium Josephinum of Columbus, Ohio, and obtained his medical degree from the Medical School of George Washington University in Washington. He did post-graduate work in eye, ear, nose and throat work in Chicago, and began the practice of medicine in 1907 at McDonald, West Virginia. He later moved to Jamestown, N. Y., where he was surgeon to the Jamestown Hospital.

Doctor Reger was a life member of the American Academy of Ophthalmology and Otolaryngology, and a member of local and state medical societies.

He is survived by his wife, two daughters and one grandson.

Alfred Edwin Rhein, M.D.

Doctor Rhein died in Terre Haute, Indiana, on October 21, 1938, at the age of fifty-five years.

He graduated from the Eclectic Medical College of Cincinnati in 1906, from the University of Illinois College of Medicine in 1913, and began to practice medicine in Rosedale, Ohio. Later he took post-graduate courses in otolaryngology, including one year in Vienna, and began the practice of his specialties in Terre Haute in 1927.

At the time of his death, Doctor Rhein was on the staff of St. Anthony's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

Doctor Rhein was very much interested in civic matters, and was an enthusiastic Mason.

His widow, one sister and two brothers survive him.

Ellison Lloyd Ross, M.D.

Doctor Ross was born in Dixon, Illinois, on June 13, 1881, and died in Waukegan, Illinois, on December 21, 1938.

He obtained his B.Sc. degree from Iowa State Collgee, Ph.D. from Illinois University, Chicago, and his medical degree from Northwestern University Medical School in 1918.

Doctor Ross was Instructor at Northwestern University Medical School from 1914 to 1918, Associate Professor of Otolaryngology from 1925 to 1928, and Professor of Otolaryngology at the time of his death. He was on the staffs of Victory Memorial and St. Therese's Hospitals in Waukegan, attending otolaryngologist to the Passavant Memorial Hospital, Chicago, and the Veterans' Administration Facility, North Chicago.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American Otological Society, and member of local and state medical societies.

He was the author of many articles on his special subjects, and intensely interested in civic matters.

His wife, one daughter and one son survive him.

Joseph James Scroggs, M.D.

Doctor Scroggs was born in Beaver, Pennsylvania, on February 3, 1877, and died there on July 19, 1938.

He graduated from Geneva College and obtained his medical degree from the University of Pennsylvania Department of Medicine in 1902, following which he interned at West Penn Hospital, Pittsburgh. He began the practice of medicine in Beaver, Pennsylvania, where his father and grandfather before him had been physicians. Later he took post-graduate instruction in otolaryngology and ophthalmology at the Mayo Clinic, and in the Clinics in Berlin and Vienna, returning to Beaver to specialize and to conduct the otolaryngological clinic at the Providence Hospital, Beaver Falls.

Doctor Scroggs was a member of the American Academy of Ophthalmology and Otolaryngology and state and local medical societies.

His widow, one brother, and one sister survive him.

Oscar Merle Shirey, M.D.

Doctor Shirey was born in Latrobe, Pennsylvania, on May 15, 1878, and died in Shaker Heights, Ohio, on November 20, 1938.

After graduating from Western Reserve University Medical Department, Cleveland, in 1903, he took post-graduate work in otolaryngology under Doctor Mosher at Harvard University, and and studied abroad at the great clinics in London and Vienna. He began the practice of medicine in Cleveland in 1905, later specializing, and at the time of his death he was on the staffs of St. Alexis, Lakeside and Polyclinic Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and local and state medical societies.

Doctor Shirey was a Captain in the World War, he was a 32° Mason, and interested in athletic and community activities.

He is survived by his wife, one son, and two brothers.

Richard Cecil Smith, M.D.

Doctor Smith was born in Norfolk, Nebraska, on March 18, 1886, and died in Superior, Wisconsin, on October 30, 1938.

Receiving his medical degree from Washington University School of Medicine, St. Louis, in 1911, he practiced medicine in St. Louis, on the Pacific Coast and in Duluth, Minnesota. In 1913 and 1914 he took post-graduate work in ophthalmology and otolaryngology in London and Vienna, and returned to Vienna for a year's study in 1923.

He began the practice of his specialties in Superior 23 years ago, and was oculist to the Great Northern Railway Company and Soo Line Railway Company. At the time of his death he was on the staff of St. Mary's, St. Francis and Good Samaritan Hospitals.

Doctor Smith was a member of the American Academy of Ophthalmology and Otolaryngology, and the Pacific Coast Oto-Ophthalmological Society, as well as local and state Medical Societies.

He is survived by his widow, three sons, his father, a sister and a brother.

Walter Hamilton Snyder, M.D.

Doctor Snyder was born in Elmhurst, Pennsylvania, on September 3, 1870, and died in Toledo, Ohio, on December 29, 1938.

His early life was spent in Scranton, Pennsylvania, and he received his medical degree in 1891 from the University of Michigan. Later he took post-graduate work in Philadelphia and New York clinics, and several European clinics, particularly in Vienna, then went to Toledo to specialize in eye, ear, nose and throat work.

Doctor Snyder was a member of the American Academy of Ophthalmology and Otolaryngology, past president of the Toledo Academy of Medicine and of the Ohio State Medical Association; and was for many years President of the Board of the Ohio Commission for the Blind. At the time of his death he was on the staffs of the Flower Hospital and the Lucas County Hospital. In 1905 he was member of the House of Delegates of the American Medical Association.

He was a militant leader for reforms and benefits to the blind, and at his death his entire estate went to the Ophthalmic Research Foundation, which he had planned for many years.

In addition to his medical work, Doctor Snyder devoted himself enthusiastically to many hobbies. He was an ardent sportsman,

and was an active member and organizer of the Black River Ranch at Onoway, Michigan, a fish and game preserve.

During the World War, he served on a Medical Advisory Committee.

Otto Jacob Stein, M.D.

Doctor Stein was born on August 8, 1867, at Council Bluffs, Iowa, and died November 21, 1938, at Los Angeles, California.

He was a graduate of Northwestern University College of Pharmacy and obtained his medical degree from Missouri Medical College in 1891, "cum laude." Later he did post-graduate work in Wurzberg, Germany. He began the practice of medicine in Chicago in 1893, and retired in 1929.

Doctor Stein was a life member of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Laryngological, Rhinological & Otological Society, as well as local and state medical societies in Illinois and the West.

Following his retirement he devoted himself to civic and cultural pursuits at Palos Verdes Estates, California.

He is survived by his wife, one daughter, and a sister.

Philip Samuel Stout, M.D.

Doctor Stout was born in Rockhill, Bucks County, Pennsylvania, on August 20th, 1877, and died in Philadelphia, Pennsylvania, on November 3, 1938.

He graduated from the University of Pennsylvania Department of Medicine in 1904, and practiced medicine there since that time, for the last quarter century specializing in eye, nose and throat work. He took post-graduate work in Vienna.

At the time of his death Doctor Stout was Director of the Department of Otolaryngology at St. Luke's & Children's Hospital, Associate Professor of Otology in the Graduate School of Medicine, University of Pennsylvania; Clinical Associate in the Department of Laryngology, Jefferson Hospital, and Clinical Associate in the Department of Rhinology and Asthma, Jefferson Hospital. He was especially interested in children and was visiting physician to the Presbyterian and Baptist Orphanages.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological & Otological Society, and of local and state Medical Societies.

In the World War he served as a lieutenant in the Army Medical Corps.

Doctor Stout's progenitors came to this country in 1700, some of them served in the Revolutionary War and one, Abraham Stout, was a delegate to the Constitutional Convention of 1780 from Bucks County. He was prominent in Masonic circles and actively interested in the Boy Scout movement.

His wife is his sole survivor.

Eugene Silas Strout, M.D.

Doctor Strout was born in Raymond, Wisconsin, on August 3, 1862, and died in Minneapolis, Minnesota, on June 25, 1939.

He received his preliminary education from the State Normal School at St. Cloud, Minnesota, and his M.D. from the Medical Department of the University of Michigan in 1891. Later he took post-graduate otolaryngological work in Chicago, London, and Vienna, and returned to Minneapolis to specialize.

He was Instructor in Ophthalmology at the University of Minnesota in 1910-1913, and at the time of his death was on the staffs of the Hillcrest and Northwestern Hospitals.

Doctor Strout was a life member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons and an Affiliate Fellow of the American Medical Association.

He is survived by his wife, two sons, one daughter, and two grandsons.

Harris Holmes Vail, M.D.

Doctor Vail was born on January 9, 1892, and died in Cincinnati, Ohio, on April 10, 1939.

He received his early education in Franklin Preparatory School, Cincinnati, his A.B. degree from Yale University in 1912, and M.D. from Harvard Medical School, Boston, in 1917, follow-

ing which he interned at Peter Bent Brigham Hospital. Later he took post-graduate work in otolaryngology at the Massachusetts Eye and Ear Infirmary.

At the time of his death, Doctor Vail was Instructor in Otolaryngology at the University of Cincinnati College of Medicine and on the staffs of the Cincinnati General Hospital, Christ Hospital, Children's Hospital, Jewish Hospital and Good Samaritan Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American Laryngological Association, and American Otological Society, and local and state medical societies.

During the World War he served as Lieutenant in the United States Navy.

The field of otolaryngology has lost a most promising man in the death of Harris Holmes Vail. Whether in technical and scientific work, the social side of the meetings or in vacation field, he was concentrated and indefatigable in his interest. He has published many praiseworthy articles and he has just received special recognition in the major senior societies of otology and laryngology.

His wife and three children survive him.

Joseph Mark Walsh, M.D.

Doctor Walsh was born in Yankton, South Dakota, on July 27, 1877, and died in Rapid City, South Dakota, on June 26, 1939.

He received his early education in Yankton College and the University of Nebraska, and his M.D. degree from the University of Illinois. In 1905 he located in Fort Pierre, later taking post-graduate instruction and specializing in eye, ear, nose and throat work in Rapid City. At the time of his death he was on the staffs of the Methodist, Deaconess and St. John's Hospitals.

Doctor Walsh was a member of the American Academy of Ophthalmology and Otolaryngology, and local and state medical societies.

He was well known for his splendid work in the fight against trachoma among the Indians in the territory around Fort Pierre. His hobbies were fishing and hunting.

His wife and five children survive him.